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Original Communications

THE OBJECTS OF DIGITALIS THERAPY*†

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NEW YORK, N. Y.

ALTHOUGH 143 years have elapsed since Withering[1] first used digitalis consistently in the treatment of heart failure, the situations in which the drug may be expected to be of benefit and the mechanism by which benefit occurs are still subjects of study and investigation.

Before discussing the subject from the present day point of view, let us see what has been the trend of knowledge regarding the use of digitalis in the past. In reviewing the literature of the use of this drug as a therapeutic agent, one finds that rather sharply defined periods are to be recognized: the following are the points of view and contributions which seem to be important.

It is to be expected that the criteria for the use of digitalis should be influenced by the notions which have been current from time to time. Withering made the first contribution[1]. Having found the drug of benefit in certain cases of edema, he advocated its general use as a diuretic. It is to be recalled that at this time Withering was unable to differentiate edema of cardiac origin from that of renal origin. This contribution was later made by Bright. Later still, Kreysig[2] expressed the opinion that digitalis contributed something to the energy of the heart, by which he probably meant that the drug increased the contractile power. He arrived at this notion from studies made in the clinic. Following these observations, the indiscriminate use of digitalis in such diseases as tuberculosis, scarlet fever, measles, and in cases of hemorrhage, caused the drug to fall into disuse. It is due to the next phase in the development of clinical physiology that it was re-established as a therapeutic agent. I refer to the discovery of the method of auscultation by Laennec[3] in 1819, followed by Hope's[4] demonstration in 1831 that closure of the valves gives rise to the heart sounds. Thus it was made possible to diagnose lesions of the

*From the Hospital of the Rockefeller Institute for Medical Research, New York.
†Read before the Section of Medicine, The New York Academy of Medicine, Dec. 18, 1928.

valves of the heart, and during the next 75 years it was in the type of valve lesion which was present that we find the criterion for the administration of digitalis. For instance its use was advocated in mitral disease and contraindicated in aortic insufficiency (Corrigan[5]). This remained the situation until 1912. Meanwhile Bouillard in 1835[6] and Traube in 1871[7] had inclined to the use of the drug as a "cardiac sedative." At the beginning of the twentieth century, Krehl[8] and Romberg[9] paved the way for a study of the significance of the behavior of muscle in heart disease, and likewise of the effect of digitalis upon the heart muscle under clinical conditions.

The next advance which exerted an influence upon this problem was the identification by Mackenzie[10] in 1911 of the rhythm of the heart known as auricular fibrillation. The striking effect of the drug in reducing the rate of the completely irregular pulse in this condition together with studies of the effect of the drug on other forms of irregular heart action led physicians to the belief that it was preëminently the irregular heart upon which digitalis acted. This view is still more or less current. So strong was it as late as 1915 that Cohn[11] emphasized the efficacy of the drug in the presence of the normal rhythm when edema was present. Attention was again directed to the action of digitalis on human heart muscle when Cohn, Fraser and Jamieson[12] in 1915 demonstrated changes in the form of the T-wave of the electrocardiogram after giving therapeutic amounts of the drug. In 1924, Dr. Cohn and I[13] showed by means of moving x-ray photographs that digitalis increased the extent of contraction of the ventricles of the human heart, in the presence of a regular rhythm as well as in the presence of auricular fibrillation.

I come next to the question of dosage and standardization. From a vast literature dealing with this phase of the subject, only one contribution of importance can be said to have emerged. I refer to the now well-established fact that large doses of digitalis may be given and that this method of administration is the one of choice. "Experience has shown that the biologic assay of the drug by the cat or by the frog method does not parallel the therapeutic effect in patients. The amount, however, of any preparation that is required to give this effect is approximately the same regardless of the age and the weight of the patient. Our experience may be of interest in this connection and illustrates the point I wish to make. We have been using for years a commercial preparation*, 1.0 gm. of which given by mouth within 24 hours may be expected to reduce the ventricular rate in rapid auricular fibrillation to the normal level, to give changes in the form of the T-wave of the electrocardiogram, and in suitable cases to induce diuresis, without the occurrence of such toxic symptoms as nausea and vomiting, and without the occurrence of ectopic

*Digitan (Merck).

beats. It is of small consequence if the effect which is desired is attained when only 0.9 gm. of the drug has been given or that it requires 0.2 or 0.3 gm. more than this amount, since the last doses need not be given or the additional amount may be given in the second 24 hours. In our experience if an effect is not obtained with approximately this amount of the preparation it is of no avail to give larger amounts. Exact information concerning the value of small or so-called "tonic" doses of digitalis is at present lacking.

Since in recent years cardiac output has been so prominent in investigations of the physiology of the circulation, it was to be expected that the effect of digitalis upon this function should be studied. The experiments which Harrison and Leonard[14] and the observations which Burwell, Neighbors and Regen[15] have recently published again focussed attention more keenly on the problem of the action of digitalis. Their observation stands out as one of the important contributions to the study of this drug. They found that soon after the administration of digitalis to normal dogs, the volume output from the heart per minute diminishes. This result was new and unexepected and ran counter to the views which have been built up in part from pharmacological studies in the laboratory and in part from inferences drawn from careful clinical observation. It had been the accepted belief that in the presence of congestive heart failure (edema) the cardiac output per minute was diminished and that improvement consisted in restoring this amount toward its former level. If this view of heart failure is correct, it appears logically to be mistaken practice to prescribe digitalis, which also decreases cardiac output. This situation, as we shall see later, should not be accepted as if all the facts involved in it were known; and we should not draw too far reaching inferences from this observation before other factors have been analyzed.

We have seen therefore in this brief review that the criteria for the use of digitalis viewed in chronological order have been: first, the presence of edema, later its effect on the heart muscle, later still, the presence of certain varieties of valvular disease, and in our own day cardiac rhythm with especial reference to auricular fibrillation, now, a return to its effect on muscle and finally, cardiac output.

In reviewing this subject recently, we were led to ask ourselves: "What are the objects of digitalis therapy?" The supreme object of all therapeutic procedures is to cure the disease completely and, failing this, to alleviate the symptoms, subjective as well as objective, from which the patient suffers. To cure is surely not the object of digitalis therapy; and the situations requiring benefit differ in the several instances in which it is used. Moreover the definition of beneficial action and how its presence is to be ascertained must be

decided. Is action to be judged in terms of an effect, such as its effect on blood pressure or volume output, or is it to be judged by its effect on tone or on contraction or on another of the many actions which it undoubtedly possesses? The answer is, Yes, if any effect goes parallel with benefit. But it does not. Benefit will have to be sought in the net result of all these, in the general reaction of the whole man.

How then, based on what is known of its physiological action, is the administration of digitalis expected to do good? We shall limit the discussion to its use in heart disease and in pneumonia.

In spite of the more or less current view that digitalis is most effective in the presence of auricular fibrillation, experience has shown that the drug is sometimes of benefit to those patients suffering from congestive heart failure in the presence of a normal or regular rhythm of the heart. What the state of the circulation is in this condition in terms of cardiac output is not known. Until recently the view was commonly held that it was decreased. Harrison and Leonard,[14] and Robinson[16] are of the opinion on the other hand that in heart failure the cardiac output may be increased. These authors have recently revived the notion first expressed by Krehl[8] that congestive heart failure is due to imbalance of the two ventricles and that digitalis rectifies this imbalance. There is no direct evidence that this is the mechanism of congestive heart failure, for such an imbalance in output must necessarily be of only short duration. Nor is there evidence for the belief that digitalis can restore balance, since the drug must act equally on the two ventricles. The mechanism by which circulatory efficiency is restored is not at present known.

As has been said, Harrison and Leonard made a significant contribution in the observation that soon after the administration of digitalis the volume output of the heart per minute diminishes. Dr. Cohn and I[17] confirmed this observation; we analyzed this effect further and were able to unravel a twofold action of digitalis. One is on tone of muscle, that is to say, it increases cardiac tone; this causes a decrease in size of the heart, from which issues a decrease in cardiac output. In other words, the heart has been made a smaller pump. A second effect is the action on contraction; the extent of the ventricular contraction is increased. This tends to increase cardiac output. The cardiac output which obtains at any instant is the net result of the working of these two opposing factors. That is to say, cardiac output is a function of size of the heart and ventricular contraction. If cardiac size is not smaller than a critical value, increase in ventricular contraction overbalances decrease in size so that cardiac output increases beyond that initial value. We have also shown that enlarged hearts in dogs without heart failure respond to digitalis in the same manner as do the hearts of normal ones.[18] This is the result one should expect. The question now naturally

arises whether these observations throw light on the way digitalis acts in heart failure in human disease. There is ample evidence in observations made in the clinic that digitalis in therapeutic amounts has an effect on the functions of tone and of contraction in the heart in man. First, with regard to tone, it is well known that an effect of digitalis upon the size of the heart can be demonstrated in heart failure by means of x-ray photographs, that is to say, a decrease in size of the heart occurs. At times this effect can be demonstrated soon after the administration of digitalis; it is however frequently not detected until after prolonged treatment with the drug. With large doses, in normal cases, the effect on cardiac output comes on soon as Burwell, Neighbors and Regen[15] showed. Though the heart does not become smaller, an influence on its size may nevertheless be demonstrated, as was shown by Levy[19] in the case of lobar pneumonia. In this disease enlargement of the heart did not occur or at least tended to take place less frequently if this drug was given. Should the mechanism of heart failure involve decrease in cardiac output, as has until recently been generally believed to be the case, point would be given to what Starling[20] described as the law of the heart. Starling showed in experiments that when heart muscle fibers increased beyond a certain optimal length, decrease in output from the heart resulted. If the optimal or somewhat shorter length were restored, output from the ventricles increased. Heart failure may be a condition in which the fibers are longer than optimal; were digitalis able to restore them to a proper length, that is to say, by exerting its effect on tone, the requirement of the situation would be met. This is the sum of our knowledge with regard to the effect of digitalis on tone. We come next to its effect on contraction. We have ourselves shown that in patients suffering from heart disease, increase in ventricular excursions may take place after the administration of digitalis in therapeutic amounts, even though no demonstrable change in the size of the heart can be seen in x-ray photographs. If increase in contraction occurs without simultaneous decrease in size of the heart, our experiments permit the inference that cardiac output increases. This may be the situation in heart failure in man, but of this there is no direct evidence. We have thought of the mechanism of recovery from heart failure as taking place in such a manner, since it is difficult for us to rationalize improvement and diuresis in congestive heart failure with decrease in cardiac output.

Because of its action in blocking auricular stimuli from reaching the ventricles, digitalis is employed in treating patients suffering from auricular fibrillation with rapid ventricular rate whether congestive heart failure is or is not present. When heart failure is not present, relief from the symptoms due to the rapid ventricular rate is to be expected. When failure is present, one is not surprised to

find that benefit occurs if failure can be ascribed to the rapid abnormal rhythm alone. Without doubt, however, its effects on tone and contraction which have already been discussed play a rôle, as in the case where the rhythm is normal. In fact we have devised experiments which show this to be the case in dogs, subjected to artificially induced auricular fibrillation.

I have shown therefore that the only actions of digitalis upon the heart which can at present be demonstrated in patients are effect on tone, effect on contraction, and effect in irregularity. If occasions arise in which these effects are desired, then the use of digitalis is indicated. I have also discussed how heart failure may be influenced by the working of these known actions.

We now come to the subject of digitalis in pneumonia. Its use in this disease at the present time is, in our experience, based on the same three readily demonstrable effects of the drug, all of which can be elicited in the presence of this disease. In the first place it is given for its effect in the presence of auricular fibrillation. During the course of pneumonia, auricular fibrillation and auricular flutter occur as complications which increase in frequency in the age groups beyond 30 years. If patients are under the influence of digitalis when auricular fibrillation begins, a great increase in ventricular rate will not occur, and the circulatory mechanism is not exposed to the strain of rapid auricular fibrillation. In the second place it is given for its effect on tone; we have already made mention of the observation of Levy[19] that the dilatation of the heart which frequently occurs during the course of pneumonia fails to occur if digitalis is administered. Although there is the possibility that dilatation of the heart in pneumonia is a compensatory mechanism, the object first mentioned is sufficiently important in our experience to warrant its being given. And lastly, in addition to its effect in auricular fibrillation and its effect on tone, it is given for its effect on contraction. Cohn has shown that digitalis induces its characteristic effects on the form of the T-wave of the electrocardiogram and on conduction time if the drug is given to patients suffering from pneumonia. If these two effects represent an effect on muscle, as they undoubtedly do, we should expect the drug also to exert its characteristic effect on contraction, that is to say, that it should increase contraction.

Harrison and Leonard[14] advise the use of digitalis in this disease on different grounds. They have shown augmented cardiac output in artificially induced pulmonary infection in dogs.[21] They infer that the cardiac output of patients suffering from pneumonia is also increased and that increased cardiac output in this disease is deleterious. They therefore advocate the use of digitalis in order to decrease the volume output from the heart. On the other hand, if increased cardiac output occurs in pneumonia, it may be a compensatory mech-

anism by which the heart responds to anoxemia. In this case to restore cardiac output to a lower level may not be desirable.

I have said that the objects of giving digitalis in cardiac disease are to increase tone, to increase contraction and to elicit its effect in the fibrillating heart, but whether these objects will be attained in terms that can be translated as benefit to an individual patient cannot be foretold. We cannot predict whether good will result from increasing tone, from increasing contraction, or from blocking the auricular impulses in a fibrillating heart. At the present time the test has to be made in each case. Not only are the conditions which regulate the effectiveness of digitalis at one time and its ineffectiveness at another time in the same patient unknown, but also the types of heart failure classified on an etiological, physiological or anatomical basis, which are likely to respond to digitalis, if there is any distinction to be made on this basis, are unknown. We no longer hold to the view current in Corrigan's time that digitalis can be prescribed on the basis of valve lesion. Marvin has recently emphasized the necessity in digitalis therapy of making distinctions on the basis of etiology. For instance, he was of the opinion that heart failure occurring in arteriosclerotic heart disease responded more frequently to treatment with digitalis than did heart failure which was the end-result of rheumatic heart disease. If this observation proves to be correct, how is such a difference to be interpreted? Is it that each of these diseases leaves a different imprint on the heart muscle, which in turn is reflected in the effect of this drug on the muscle? Although digitalis will still give changes in the T-wave of the electrocardiogram that are similar in both instances, the effect on tone and on contraction may be attenuated.

We have seen that the administration of digitalis is still far from being a simple problem. If an irregularity of the heart needs to be controlled, if tone of the muscle needs to be increased, if contraction of the heart needs to be strengthened, then the use of digitalis is indicated. It remains for future study to ascertain precisely how benefit is to be recognized, how benefit occurs, how benefit is to be measured and in what instances benefit is to be expected.

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THE NERVOUS HEART*

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IT IS fashionable at meetings of cardiologists to discuss various aspects of abnormal structure or function of the circulatory apparatus. I have turned aside and chosen as subject the "nervous heart" for three practical reasons: first, because this is one of the most common if not the most common condition the cardiologist sees; second, because it is the one in which therapy can be most brilliant; and third, because unfortunately it is also the condition most often receiving poor treatment.

The term nervous heart is of course not scientific; but it does serve the useful purpose of designating a group of disturbances in the efferent or afferent (often both) nerves of the heart, caused in many ways, and of which the essential feature in most instances is an anxiety neurosis of greater or lesser severity. The efferent disturbances affect the frequency or force of the heart action, the afferent result in heightened perception of the heart action, pain or other abnormal sensation. Both phenomena commonly occur together through interaction with the mind. For example, there may be increased heart action resulting in increased afferent stimuli and hence mental disturbances; or heightened heart consciousness may cause mental disquietude and thence increased heart action; or fear may initiate the vicious circle.

Etiology.—Both sexes and all ages beyond early childhood are affected, but more commonly young adults and women at the menopause. Sedentary occupations contribute relatively more cases, probably because they attract the less rugged elements of the population. The hereditary neurotic type of constitution is the important predisposing cause. Many such subjects are dextrosinistrals or "cross wired," i.e., if naturally right handed will aim a gun with the left eye or vice versa—a very interesting personality type described by Quinan.[1]

The condition is precipitated by a large variety of factors in the heart itself, in the rest of the body or in the mind. Crippled valves, abnormal heartbeat mechanism and hypertension do not protect against neurosis, but on the contrary may be the starting point for purely neurotic symptoms which are often more important than the organic changes present—important in the practical sense of producing greater suffering and disability and offering greater possibilities for treatment. This is a fact which is inadequately appreciated. All practitioners of medicine, but especially those who treat heart cases, should have at

*Read at the annual scientific session of the American Heart Association, Portland, Oregon, July 9, 1929.

[1]Quinan, C.: Arch. Neurol. and Psych. 7: 352, 1922, and personal communications.

least a superficial acquaintance with the anxiety neurosis, including its formes frustes, and some aptitude for its prophylaxis and treatment.

Precipitating factors in the body outside the circulatory apparatus are in general those which affect adversely general strength, sleep, weight, and quality of blood and other tissues. Many cases appear during convalescence from infections or in the presence of focal infections, after prolonged or excessive effort, etc. Others are associated with disturbance of the gastro-intestinal tract or the ductless glands, especially the thyroid. Tea, coffee, and tobacco in adequate dosage in susceptible persons undoubtedly do produce irritability of the heart, i.e., acceleration, a "bumpy" action, premature contractions, etc., with or without heightened heart consciousness; but such direct effects have been much overrated, and when these substances are effective, it is more often indirectly through interfering with nutrition, sleep, etc. What has been loosely termed the "cigarette heart," cases of the sort we are discussing, constituted the bulk of those referred from the British army to their heart hospital during the war; and analysis showed that, whereas these men were culled from an army of almost universally excessive smokers, they themselves for the most part smoked little or not at all. This finding, of course, is not to be construed as an argument for the protective influence of tobacco, but simply as another testimony to the fact that certain types of persons tend to develop cardiac irritability—the same types that are likely to eschew tobacco and adopt diet fads.

But the most important precipitants are psychogenic. Mental processes, conscious or subconscious, interplay with most, perhaps with all other etiological factors, and are sometimes solely responsible. Many cases develop after business reverses, domestic maladjustment and the like. Modern civilization provides a fertile soil for the growth of psychoneuroses and especially those concerned with the heart. I refer not especially to the familiar "pace at which we live," but rather to the general diffusion of knowledge about the vital function and the diseases of the heart. Valvular disease, high blood pressure and angina pectoris are common fireside topics. Organizations with philanthropic and educational aims such as the American Heart Association stress in the mind of the public the prevalence of heart disease. Such publicity is of course necessary, and I do not criticize the work of this Association; but I do urge that all who have contact with the public through the printed page, the lecture platform or the clinics keep constantly in mind the danger and do the utmost possible to avoid it. It should be emphasized before the public that most "heart symptoms" do not mean heart disease at all, and that those who think they feel them should promptly transfer the responsibility to their medical advisors.

This is the best that can be done, and yet we may as well frankly

admit that the long-suffering common man will often encounter further grief when he follows this advice. For the average medical practitioner still has much to be desired in ability to discriminate between important and unimportant heart signs and symptoms. With a dim understanding of precordial pain, irregular heart action and murmurs, the physician who encounters any such phenomena, even in a patient he believes to have a sound heart will usually add, after a feeble reassurance, the advice to "be careful"; and this may be all that is needed to confirm an incipient neurosis. Or the neurosis may be entirely fabricated out of contact with the doctor, as the following case illustrates:

A young male bookkeeper was found to have a faint functional systolic murmur and frequent ventricular premature contractions. The latter were identified only by careful auscultation and by electrocardiogram. The remainder of the examination, including x-ray of heart, was entirely negative. Symptoms were extreme heart consciousness especially of the "big bump" following the premature contractions, frequent lancinating precordial pain, breathlessness on effort and fatigue all the time, all of which had resulted in idleness for six months and bed rest much of the time. The interesting feature was the onset six months previously. At that time he had been rejected for employment at a hospital because the examining physician had found "heart-block and a heart murmur." The doctor evidently had been vastly interested in his discovery, for he had called one or two other doctors to see it; and after free discussion in the presence of the amazed applicant, they had declined to take him as an employee but graciously accepted him as a patient and put him to bed. The patient admitted that all his symptoms dated from that day and that prior to that time he had led an active life and had never known that he had a heart.

Gross errors in diagnosis are not the only things to be avoided. More common is the overestimation of the gravity of a compensated valvular disease or hypertension and the imposition of ill-advised restrictions in work or recreation. Or again, after a competent internist or cardiologist has made a correct estimate of the physical state of the circulatory apparatus the patient will misinterpret his words or even his silence. I have heretofore described the lawyer who knew that he had hypertension (a very benign hypertension), and who left the consulting room of a new doctor in a state of panic because in testing blood pressure the doctor said nothing but looked wise. That doctor undoubtedly enjoyed the agreeable sensation of psychological devoir skillfully performed. He had carefully avoided uttering any alarming words, and remained naïvely ignorant of the fantastic interpretation which the penetrating lawyer had derived from that wise look. Reflection over this case has given me what I believe is a definite refinement in blood pressure technic. It is to cultivate at the moment of observation a bored expression rather than a wise one; and then if I wish not to announce the reading, to inquire, while the air is yet sissling out of the machine, "How are your bowels?"

SYMPTOMS

Nervous heart symptoms include many of those of organic heart disease and, as has been suggested, are often superadded to heart disease. Anxiety is the most important and usually stands in a mixed relation of cause and effect with the subjective and objective heart phenomena. Among the sensory experiences of patients, heightened consciousness of heart action is most prominent. Usually referred to the precordium, the pulsatile sensation may be complained of in the ears, the neck, epigastrium or extremities. In mild cases, it may be simply an exaggeration of the normal heart action perception associated with effort or excitement; and these patients may find effort limited by this sensation rather than ordinary hyperpnea. Or it may be only the "bump" following a premature contraction which is felt or the regular heartbeats after using tea, coffee or tobacco, or after meals, or on lying down, or when in an elevated altitude. In more severe cases the pulsations are felt under all circumstances.

Often associated with heart consciousness, but at times independent, is the symptom of position aversion. Most normal persons and many with various types of heart disease can lie comfortably on the back or either side. Cardioneurotic patients may have no special preference, but as a rule they do. They object most often to lying on the left side, sometimes the right side or either side or occasionally the back. As reason they describe vague discomfort, increased heart consciousness, smothering sensation or precordial pain. Only occasionally do uncomplicated cases require more than one pillow. That this position aversion as well as heart consciousness in general is a sensory phenomenon rather than due primarily to change in cardiodynamics is suggested by the fact that so many patients with crippled or very large hearts do not show it. One young man with a "cor bovinum" due to aortic insufficiency said that he avoided lying on the left side, not because it gave him the slightest discomfort, but because the action of his heart in that position rocked the bed so violently that his wife could not sleep!

Precordial pain is common. It rarely has the compression quality suggestive of angina pectoris but is usually a dull ache, sore feeling, burning sensation, or a lancinating pain. The latter is occasionally severe, like a knife-thrust, a stroke of lightning, etc., and is often confused with angina. These pains rarely have any close relation to effort, excitement, meals, etc. They may radiate to the left arm or elsewhere. With or without pain there is often precordial sensitiveness to touch, so much so that patients sometimes try to avoid ordinary contact of clothing over the heart, especially the region of the apex.

Other common symptoms are the group associated with the effort syndrome: easy fatigue, breathlessness with slight effort (sometimes failure to obtain satisfaction from a deep breath, without exercise),

increased sweating, especially of the axillae and hands, faintness, tachycardia, and cool, moist and often cyanotic extremities.

PHYSICAL EXAMINATION

Physical examination reveals nothing specifically indicating the nervous heart, for it must be remembered that it occurs in those with diseased as well as normal hearts; and the effort should be rather to detect nervous and especially psychic deviations from normal. In the effort syndrome group will be found the usual tachycardia, poor response to effort, etc. Search should be made for underlying causes such as constitutional inferiority, glandular disturbances and infections.

TREATMENT

Treatment is first of all prophylactic. Remembering the ease with which grave psychic traumata may be inflicted, the intelligent and conscientious doctor will spare no pains to exhibit a salutary conversation and demeanor before the sort of patient who is a candidate for neurosis. He will not as a rule conceal abnormal findings, because the denouement which is likely to follow at a later date will probably be a worse blow than the properly understood facts at the outset. In order to help these patients the doctor must above all things have their confidence, and this will be denied him unless he establishes a reputation for candor. And the plain facts properly understood (and so necessary to be understood if the cardiac patient is to care for himself properly) are rarely so damaging psychologically as the fantastic mental pictures of the patient who feels that his physician is concealing things.

The war experience emphasized the importance of slow resumption of work after infectious diseases and also the frequency of cardiac irritability when men naturally fitted for and habituated to clerking were subjected to heavy physical strain. These lessons should be remembered in civil practice.

For some of the established cases of neurosis, technical psychotherapeutic methods in the hands of experts may be needed. But these are few compared with the great numbers of mild cases where simple encouragement and a suitable regimen, usually involving gradual resumption of activities, are sufficient. Some patients need coaching over a period of time. For others a single interview will effect a cure; but the examination should be extensive enough to make the patient feel that his condition has received adequate investigation. From this point of view x-rays and electrocardiograms are often legitimate therapeutic adjuncts even when they are not considered essential to diagnosis.

(For discussion, see page 115.)

STUDY OF T-WAVE NEGATIVITY IN PREDOMINANT VENTRICULAR STRAIN*

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IT HAS been known for a long time that certain cardiac lesions throw a burden chiefly on the right or on the left ventricle. This has been manifested by the finding at necropsy of hypertrophy and dilatation predominantly of one or the other ventricle. Clinicians are familiar with the evidence of failure of the right or of the left side of the heart: hepatic engorgement and edema of dependent members when the right ventricle has failed, and marked dyspnea and pulmonary edema when the left ventricle has failed.

Early in electrocardiographic studies, certain modifications of the QRS complexes came under careful scrutiny in relation to anatomical preponderance of the right and of the left ventricles. The work of Einthoven, Lewis,[14] Herrmann and Wilson, Pardee,[20] and others, has shown how closely such electrocardiographic changes parallel the actual changes in the size of the right and of the left ventricles. Lewis[15] has summarized the limitations that attend the attempt to evaluate on the basis of electrocardiographic evidence the changes in mass of the right and of the left ventricles that are indicative of preponderance. It is of interest to the present discussion to state that such correlations have a fairly high degree of accuracy when certain exceptions are borne in mind.

Nowhere has the idea been clearly advanced that a relationship may be observed between types of inversions of the T-wave and injury predominantly to the right or to the left ventricle in man. Herrmann and Wilson pointed out that ventricular preponderance does not produce characteristic changes in the T-wave. They suggested, however, that modifications of the T-wave occurring in hypertrophic preponderance of one or the other ventricle may be the result of myocardial changes which accompany most forms of heart disease.

Interest in the study of significant alterations in the T-wave was stimulated by the work of Willius,[26, 28] who showed their importance in relation to prognosis. He found that among patients whose electrocardiograms exhibited significant inversions of the T-wave mortality was double or treble that among comparable cases in which inversion of the T-wave did not occur. In his series of seventy-four patients with hypertension, whose electrocardiograms showed inversion of the T-waves in Lead I or in Leads I and II, the mortality ranged from 70 to 80 per cent, and death occurred on the average in a little less than

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eleven months. In comparable patients of the same sex and age, but without inversions of the T-wave, the mortality varied from 15 to 31 per cent, and death occurred on the average in a little less than thirteen months. In twenty-eight cases[31] of aortic regurgitation in which electrocardiograms showed inversion of the T-wave in Lead I or in Leads I and II, the mortality was 66 per cent as compared with a mortality of approximately 30 per cent in cases with a similar condition but without inversion of the T-waves. Further observations in these and in other cardiac diseases[9, 16, 29, 31] have shown that mortality is greater among those cases in which there is inversion of the T-wave, as compared with those in which such electrocardiagraphic changes are not observed.

In the daily clinical observation of patients suffering with cardiac lesions which are capable of producing a differential effect on the two ventricles, we have observed certain correlations between the type of inversion of the T-wave and that of the ventricle chiefly affected. In particular, we have been impressed clinically with the fact that in lesions which chiefly affect the left ventricle, inversions of the T-wave, when they occur, usually are found in Lead I or in Leads I and II, whereas in lesions in which the load is thrown predominantly on the right ventricle, inversions of the T-wave, when present, are found chiefly in Leads II and III. This observation led to a more detailed examination of the present material to ascertain the degree of accuracy of this observation.

In the present study, cases were chosen in which electrocardiograms had shown significant inversions of the T-wave* and which presented evidence of pathological changes capable of exerting a differential strain on the ventricles. It was found that only cases in which a detailed pathological study had been made were suitable for this study. This is due to the fact that clinical methods, although usually sufficient to disclose the main lesions of cardiac disease, may fail to identify all the pathological changes that have a bearing on the question of which ventricle is chiefly overburdened. A few cases in which there was inversion of the T-wave were excluded because pathological evidence of cardiac lesions was absent and therefore there was no basis in these cases for predicating more strain on one ventricle than on the other. This group was composed largely of cases of hyperthyroidism.

Definite Hypertension.—There were forty-two patients with definite hypertension and without other cardiac lesions which would modify the effect of the hypertension on the left ventricle (Table I). The average age of the patients was fifty-two and four-tenths years. The average blood pressure in millimeters of mercury was 202 systolic and 131 diastolic. The average cardiac weight exceeded the estimated weight

*Inversion of the T-wave in Lead III alone is not considered significant, and cases in which there were inversions of the T-wave in this lead only, were not included.

TABLE I

CASES OF DEFINITE HYPERTENSION WITH T-WAVE NEGATIVITY

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
5	44 M	211	139	+	Left	Markedly enlarged	422	Negative	Marked hydropericardium	0	0	Extrarenal sclerosis; probably chronic glomerulonephritis	No digitalis given
10	84 M	220	164	(1)* +	None	618	305	Negative	Acute fibrinous pericarditis	1+	1+	Arteriolar sclerotic atrophy of kidneys	No digitalis given; second and third electrocardiograms, taken two months later, showed no T-wave changes; hypertrophy and dilatation of left ventricle 2+; hypertrophy of right 1+; malignant hypertension
14	52 M	135	91	+	None	Much enlarged		Dilatation, of aortic, mitral, and tricuspid rings	Negative				No digitalis given; auricular flutter with 2:1 block; left ventricle markedly thickened, right very thin; marked dilatation of all heart chambers; fatty changes in myocardium
20	28 F	180	150	+	Left	Much enlarged	288	Negative	Negative	0	0	Chronic diffuse nephritis; discs blurred; low grade optic neuritis	No digitalis given; marked dilatation of heart; marked fatty degeneration of myocardium
21	61 M	204	114	+	Left	700		Negative	Negative	1+	1+	Hemorrhages in brain; right hemiplegia day before death; arteriosclerotic atrophy of kidney	No digitalis given; hypertrophy 3+ and dilatation 2+ of both ventricles

*Numeral in parenthesis signifies the electrocardiogram.

TABLE I—CONT'D

23	42 M	240	140	+	Left	573	300	Slight sclerosis of mitral and aortic	Slight thickening over right auricle	2+	2+	Chronic diffuse nephritis	No digitalis given; both ventricles hypertrophied; aortic sclerosis 2+
31	60 M	220	132	+	Left	524	300	Negative	Negative	2+	2+	Arteriosclerotic atrophy of kidneys; arteriosclerosis 2+ with retinitis 2+ of malignant hypertensive type	No digitalis given; left ventricle hypertrophied 3+, right 2+; left auricle hypertrophied 1+; malignant hypertension
32	50 F	168	120	+	Left	525		Sclerosis 3+ of mitral	Negative	3+	3+		No digitalis given; exophthalmic goiter also slight myocardial fibrosis; no definite infarction
34	49 F	220	138	+	Left	1.5 times normal size	244	Aortic and mitral leaflets slightly increased in thickness	Negative	1+	1+	Advanced renal arteriosclerosis; marked albuminuric retinitis	No digitalis given
45	75 F	154	107		Left	550	275	Negative	Negative	1+	1+		First electrocardiogram showed diphasic T in Lead I; 22.5 c.c. digitalis before second electrocardiogram, which showed diphasic T in Lead I and inverted T in Leads II and III; both ventricles dilated
53	60 F	160	107	+	Left	620	354	Negative	Negative	1+	1+	Advanced arteriosclerosis of brain with cerebral softening; advanced arteriosclerosis of kidney; fundus oculi negative	Eight c.c. digitalis before first electrocardiogram; hypertrophy of left ventricle; dilatation of both auricles; complete right bundle-branch block
55	68 F	220	138	+	Left	905	414	Negative	Negative	2+	2+	Renal arteriosclerosis 3+; retinal sclerosis of hypertensive type	No digitalis given; uniform hypertrophy of ventricles

TABLE I—CONT'D

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
60	48 M	195	130	+	Left	705	280	Negative	Negative	3+	3+	Marked thickening of renal vessels; retinal sclerosis 3+	No digitalis given
68	59 M	260	150	+	Left	680	377	Negative	Negative	2+	2+	Arteriosclerotic kidneys; marked retinal arteriosclerosis	No digitalis given; left ventricle markedly dilated and hypertrophied 4+
71	60 M	142	104	+	Left	730	428	Negative	Negative	1+	1+		Eight c.c. digitalis before electrocardiogram; hypertrophy and dilatation of left ventricle 2+
72	45 M	228	140	+	Left	750	422	Negative	Negative	2+	2+	Arteriosclerotic kidneys; retinal changes of malignant hypertension	Four c.c. digitalis before electrocardiogram; markedly thickened left ventricle.
78	40 F	250	130	+	Left	565		Negative	Negative	2+	2+		No digitalis given; left ventricle wall 2 cm. in thickness
81	35 M	200	150	+	None	490	278	Negative	Acute fibrinous pericarditis (terminal)	2+	2+	Chronic nephritis with thickened vessel walls; retinitis of nephritis	No digitalis given
85	63 M	175	120	+	Left	807	300	Negative	Negative	1+	1+		No digitalis given; hypertrophy 2+ and dilatation of all chambers

TABLE I—CONT'D

91	58 M	203	111		Right Ch'gd to Left	348	353	Slight arteriosclerotic puckering of mitral valve	Negative	2+	2+	Mild retinal arteriosclerosis of hypertensive type	First electrocardiogram normal; 18 c.c. digitalis two months before second electrocardiogram (further digitalis?); second and third electrocardiograms showed T-wave inversion in Lead III, fourth showed diphasic T in Lead I; moderate hypertrophy of left ventricle; anginal symptoms; some fibrous replacement of muscle in posterior surface of left ventricle
97	62 M	150	110	+	Left	648	363	Negative	Negative	2+	2+		First electrocardiogram showed inverted T in Leads I and II, second showed diphasic T in Lead III, third showed inverted T in Leads III (last 2 were taken after 18 c.c. digitalis, during digitalis intoxication); mural thrombosis left auricle; hypertrophy of both ventricles; marked dilatation of right auricle and ventricle
101	64 M	260	180	+	Left	825	490	Negative	Negative	2+	2+	Arteriosclerotic atrophy of renal vessels	Electrocardiogram after 4 c.c. digitalis; hypertrophy and dilatation of left ventricle
102	66 M	180	105	+	Left	845	451	Negative	Negative	1+	1+		Electrocardiogram after 6 c.c. digitalis; right bundle-branch block; QRS = 0.18 second; markedly dilated left ventricle; paroxysmal dyspnea
103	50 M	220	140	+	Left	623	323	Negative	Pericardium thickened but not obliterated (terminal)	2+	2+	Arteriosclerotic renal vessels; retinal arteries much reduced in caliber with edema of discs 2+	No digitalis given; left ventricle 3 cm. thick, right 1.7 cm.; malignant hypertension
110	55 F	255	150	(2+)	Left	516	396	Negative	Negative	1+	1+		Some digitalis taken before first electrocardiogram; normal T-waves in first, 10.5 c.c. digitalis before second; arborization block; QRS = 0.16 second; paroxysmal dyspnea

TABLE I—CONT'D

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
121	36 F	230	160	+	Left	415	284	Negative	Negative	2+	1+	Retinal sclerosis; second stage hypertension	No digitalis given; malignant hypertension; thrombi in auricles and apex of left ventricle
124	54 M	250	150		None	452	271	Negative	Negative	2+	2+	Marked thickening of renal vessel walls	No digitalis given; T-wave inverted in Leads I, II, and III
126	42 M	152	109	+	Left	792	343	Slight thickening of mitral valve (atheroma)	Small area of acute fibrinous pericarditis	1+	1+		No digitalis given; both pleural cavities obliterated; adhesions of pericardium with diaphragm; incomplete bundle-branch block; QRS = 0.11 second
131	29 F	210	120	+	Left	450	196	Negative	Negative	0	0	Renal arteriosclerosis; retinal arteries small but not sclerosed; retinitis of chronic nephritis	No digitalis given
139	52 F	180	120	+	None	366	245	Negative	Negative	1+	1+	Greatly thickened renal vessels; slight retinal arteriosclerosis	No digitalis given; marked dilatation of right ventricle with mural thrombosis
140	37 F	254	172		None	540	217	Negative	Negative	1+	1+	Marked renal arteriosclerotic thickening; stage 3 of malignant hypertension with retinitis 3+	No digitalis given; T-wave inverted in Leads I, II, and III; moderate hypertrophy of left ventricle

TABLE I—CONT'D

143	48 M	180	140	+	None	403	225	Negative	Old adhesive tags anterior surface (slight adhesive pericarditis)	1+	1+	Arteriosclerosis with softening of the central nervous system; retinitis of essential hypertension or nephritic type	No digitalis given; dilatation 3+ of left ventricle and 2+ of right; blood urea 224
145	47 M	200	128	+	Left	820	371	Negative	Negative	1+	1+	Marked fibrosis of renal vessels; retinitis of malignant hypertensive type graded 2-3	No digitalis given; dilatation of left ventricle
147	65 F	165	120	+	Left	684	300	Negative	Negative	2+	2+	Renal blood vessel walls thickened	Seven and five-tenths c.c. digitalis before first electrocardiogram; complete right bundle-branch block; hypertrophy chiefly of left ventricle
158	22 M	190	156	+	Right	474	300	Right coronary and noncoronary cusps are bound together with thickening of commissures	Negative	1+	1+	Syphilis of the central nervous system; sclerosis; cerebral hemorrhage; renal arteriolar sclerosis; retinitis of malignant hypertensive stage 2-3	No digitalis given
165	45 M	204	164	+	None	505	300	Negative	Negative	1+	1+	Marked sclerosis of renal vessels; retinitis 2+; malignant hypertension	Eight c.c. digitalis before electrocardiogram; hypertrophy and dilatation of left ventricle
166	52 M	228	150	+	Left	700	325	Negative	Negative	2+	2+	Renal arteriosclerotic atrophy; retinal arteriosclerosis 3+	No digitalis given; hypertrophy of both ventricles but chiefly of left

TABLE I—CONT'D

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
167	39 F	250	160	+	Left	435	250	Mitral valve thickened slightly with a little puckering and a pinpoint hemorrhage	Negative	1+	1+	Renal arteriosclerosis; retinal arteries irregularly contracted	No digitalis given
168	56 M	155	105	+	Left	474	300	Fusion of commissures of aortic valve	Negative	2+	2+	Reduced caliber of retinal vessels	No digitalis given
169	64 F	230	110	+	Left	435	275	Negative	Negative	2+	2+	Encephalomalacia; primary contracted kidney; retinal arteriosclerosis 1+	No digitalis given; first electrocardiogram showed diphasic T in Lead I, second inverted T in Lead I-II
177	70 M	180	90	+	Left	760	375	Negative	Negative	1+	1+		Four and five-tenths c.c. digitalis before electrocardiogram; complete right bundle-branch block; QRS = 0.14 to 0.16 second; hypertrophy 3+ of left ventricle and 2+ of right; dilatation 2+ of right ventricle
182	67 M	160	80	+	Left	712	373	Negative	Negative	1+	1+	Enlarged kidneys	No digitalis given; hypertrophy of both ventricles

of the normal heart by 280 gm.* In thirty-eight cases (90.5 per cent) the T-wave was inverted in Lead I or in Leads I and II. In two cases the T-wave was inverted in Leads II and III, and in each of these the T-wave was diphasic in Lead I.

In one of these two cases there were four electrocardiographic tracings, only one of which showed significant inversion of the T-wave. Eighteen cubic centimeters of the tincture of digitalis was given before the second electrocardiogram was made, but it could not be determined whether further digitalis was given before the fourth and significant tracing. In the other case the inversion of the T-wave in Leads II and III followed treatment with digitalis.

Certain observations to be made later in this study, together with observation of electrocardiographic changes observed in patients to whom digitalis is being administered, strongly suggest that this drug has a tendency to cause inversion of the T-waves in derivations II and III, a change which, as we shall observe later, is the same as that observed in conditions producing strain predominantly on the right side of the heart. In two cases, the T-wave was inverted in Leads I, II, and III. This group may include certain cases in which the inversion in Lead III is not of abnormal significance; if so the significant inversion is that in Leads I and II. Repeated electrocardiographic tracings often will show that the T-waves are not actually inverted in all leads and that the significant inversion is either in Leads I and II or in Leads II and III.

Probable Preexistent Hypertension.—Thirteen patients were classified as probably having had hypertension (Table II). This classification was determined largely on the basis of the size of the heart, evidence of previous cerebral vascular accidents, presence of abnormalities in the ocular fundus indicative of arteriosclerosis or of the occurrence of marked renal vascular injury in cases in which determinations of blood pressure indicative of definite hypertension were lacking. The average age of the patients was fifty-eight and two-tenths years. The average blood pressure was 139 mm. systolic and 83 mm. diastolic. The weight of twelve hearts was known, the average of which was 607 gm. The average cardiac weight exceeded the estimated normal cardiac weight by 258 gm. The T-wave was inverted in Lead I or in Leads I and II in thirteen (100 per cent) of the cases.

From a study of the combined group of patients, with definite or probable preexistent hypertension, it was observed that inversions of the T-wave in Lead I or in Leads I and II occurred in 93 per cent.

Definite Hypertension With Marked Coronary Sclerosis.—The condition in nine patients was classified as definite hypertension with marked coronary sclerosis (Table III). The average age of the patients was

*The estimated normal cardiac weight was calculated according to the tables prepared by Smith.[25]

Table II

Cases of Probable Preexistent Hypertension With T-Wave Negativity

Case	Age and Sex	Average Blood Pressure		T-Wave Inversion Leads I or I-II	Ventricular Preponderance	Weight of Heart, Gm.		Valves	Pericardium	Coronary Sclerosis		Significant Cerebral, Renal, and Retinal Data	Miscellaneous
		Systolic	Diastolic			At Necropsy	Estimated Normal			Right	Left		
1	57 F	170	60	+	Left	515	405	One calcified plaque in aortic cusp	Negative	1+	1+	Hemiplegia; sclerosis 2+ of cerebral vessels; fundus oculi negative	Digitalis eight days before coming to the clinic; aorta sclerosed 2+; exophthalmic goiter; diagnosis of probable hypertension based on heart weight; stroke 3 years ago, and twenty-four hours before death
26	56 M	120	95	+	Left	Marked enlargement		Negative	Negative			Recently formed white infarction of the kidney	No digitalis given; right bundle-branch block; size of heart suggestive of hypertension; marked hypertrophy of left ventricle; marked dilatation of all heart chambers and valve rings
50	51 M	120	98	+	Left	682	300	Negative	Slight adhesions	0	0		No digitalis given; dilatation of left ventricle
58	52 F	191	94	+	Left	500	276	Small vegetations; aortic valve 1 mm. in diameter	Negative	1+	1+	Renal blood vessels thickened 3+; considerable consecutive arteriosclerosis of the retinal vessels	No digitalis given; marked hypertrophy of left ventricle; aortic sclerosis 3+
76	73 F	180	94	+	Left	366	288	Thickening of medial leaf of mitral valve; no stenosis	Negative	2+	2+	Reduction in caliber of arteries and senile fibrosis of the fundus oculi	No digitalis given; no myocardial fibrosis except a little in anterior papillary muscle of left ventricle; moderate thickening of wall of left ventricle

TABLE II—CONT'D

79	63 F	135	95	+	Left	400		Acute terminal mitral endocarditis	Acute terminal pericarditis				No digitalis given; probable hypertension based on heart size and on absence of pericardial adhesions and enough mitral disease; blood pressure also suggestive; left ventricle dilated 3+
80	54 M	140	80	+	Right	570	451	Negative	Negative	1+	1+		No digitalis given; diagnosis on basis of heart size and absence of coronary sclerosis or valve or pericardial lesion; all heart chambers dilated 3+
111	68 M	102	70 (?)	+	Left	922	350	Negative	Negative	0	0	Narrowing and occlusion of renal arteries	Two c.c. of digitalis before electrocardiogram; left ventricle especially dilated
132	57 M	112	85	+	Left	657	270	Negative	Negative	2+	2+	Contracted scarred kidney; extensive arteriosclerosis	No digitalis given
144	59 M	170	88	+	Left	545	325	Negative	Negative	2+	2+	Sclerosis 3+ of renal vessels; sclerosis 2+ of retinal arteries; hemorrhagic type of retinitis	No digitalis given
172	46 M	110	95	+	Left	784	300	Mitral negative	Pericarditis over right auricle	2+	2+		Ten and five-tenths c.c. digitalis before first electrocardiogram; incomplete bundle-branch block; relatives said patient had previous history of hypertension; hypertrophy and dilatation of both ventricles and left auricle
176	54 M	128	58	+	Left	656	500	Negative	Negative	2+	2+		No digitalis given
178	67 M	140	65	+	None	685	377	Negative	Negative	1+	1+	Sclerosis 1+ of retinal arteries	No digitalis given; blood urea 164; hypertrophy and dilatation 2+ of left ventricle and 1+ of right

Table III

Cases of Definite Hypertension and Definite Coronary Sclerosis

Case	Age and Sex	Average Blood Pressure		T-wave inversion Leads I or I-II	Ventricular preponderance	Weight of Heart, Gm.		Valves	Pericardium	Coronary Sclerosis		Significant Renal and Retinal Data	Miscellaneous
		Systolic	Diastolic			At Necropsy	Estimated Normal			Right	Left		
13	69 M	144	80	+	Left	522	369	Arteriosclerosis 1+ of aortic; no fusion of valve cusps	Negative	4+	4+	Sclerosis 1-2 of retinal and choroidal arteries	No digitalis given; auricles dilated 2+; paroxysmal dyspnea; slight fibrous streaking of myocardium
19	67 M	184	86	+	Left	646	382	Slight arteriosclerotic changes valves	Negative	3+	3+	Pyonephrosis with stones; examination of the fundus oculi not satisfactory	Eight c.c. digitalis before electrocardiogram; slight fibrous streaking; no definite infarction; sclerosis 3+ base of aorta; also had elevated urea and low renal function; diffuse cardiac hypertrophy
25	61 M	179	125		Left	Markedly enlarged		Fibrous thickening of aortic and mitral leaflets	Negative	3+	3+	Fundus oculi negative	No digitalis given; T-wave inverted in Leads I, II, and III; left ventricle twice normal thickness
34	62 F	240	100	+	Left	Markedly enlarged		Slight fibrous change in mitral and aortic valves	Negative	3+	3+	Walls of the renal vessels thickened; negative fundus oculi	No digitalis given; complete auriculoventricular dissociation

TABLE III—CONT'D

56	60 M	170	110	+	Left	700		Thickening along line of closure of aortic and mitral valves	Negative	3+	3+	Marked thickening of renal vessels	No digitalis given; hypertrophy and dilatation of left ventricle
114	73 M	180	100	+	Left	565	284	Sclerotic aortic valve	Negative	3+	3+	Slight senile arteriosclerosis of fundus	No digitalis given; third electrocardiogram showed T-wave inversion in Leads I or I-II; as did second and fifth in Leads I-II-III; no T-wave changes in first and fourth; nodal tachycardia preceded last tracing
134	56 F	220	124	+	Left	672	351	A little atheroma aortic and mitral	Negative	2+	2+	Arteriosclerotic changes in the kidney; left homonymous hemianopia	No digitalis given; thrombus in left auricular appendage; angina pectoris clinically; diffuse fine fibrous streaking throughout left ventricle
173	59 F	165	115	+	Left	402	275	Negative	Negative	3+	3+	Retinal sclerosis 2+ of hypertensive type	No digitalis given; both coronaries practically occluded in places; clinically angina pectoris; occasional fibrous streaking of myocardium
183	52 M	154	102	+	Left			Not reported	Negative	3+	3+		No digitalis given; hypertrophy and dilatation of left ventricle; incomplete bundle-branch block

sixty-two years. The average blood pressure was 182 mm. systolic and 105 mm. diastolic. The average cardiac weight in six patients was 584 gm., which exceeded the estimated normal cardiac weight by 252 gm. In eight cases, 88 per cent, inversions of the T-wave in Lead I or in Leads I and II occurred. In one case, at times there were upright T-waves and at other times inversion of the T-wave in all leads. At least one of the tracings in which there was inversion of the T-wave was taken following a paroxysm of nodal tachycardia, and the influence of such an occurrence cannot be positively excluded in the other tracings in which the T-waves were inverted. In the electrocardiogram of one patient there was inversion of the T-wave in all leads.

Probable Preexistent Hypertension With Marked Coronary Sclerosis.—Six patients were grouped as probably having preexistent hypertension with marked coronary sclerosis (Table IV). The average age of the patients in this group was sixty-one and five-tenths years. The average blood pressure was 143 mm. systolic and 86 mm. diastolic. The average cardiac weight was 478 gm., which exceeded the estimated normal cardiac weight by 221 gm. The T-wave was inverted in Lead I or in Leads I and II in five cases (83.3 per cent). In one case, the T-wave was inverted in all leads, but the facts that this phenomenon was preceded by administration of much digitalis and that hitherto the electrocardiogram had been normal indicate that the inversion was produced by digitalis.

In the combined group of patients with definite or probable preexistent hypertension, when this was complicated by marked coronary sclerosis, the T-wave was inverted in Leads I or in Leads I and II in 86.6 per cent of the cases. Comparing this with the group of patients with definite or probable hypertension, without definite coronary sclerosis, it is found that the same changes in the T-wave occur in both groups. Hypertension when accompanied by coronary sclerosis produces changes in the T-wave similar to those found in hypertension alone, unless myocardial infarction is also present.

Syphilitic Aortic Insufficiency.—Eight patients who had syphilitic aortitis with aortic insufficiency had significant inversions of the T-wave (Table V). The average age of the patients was forty-four years. The average blood pressure was 138 mm. systolic and 45 mm. diastolic. The average cardiac weight in five cases was 620 gm., which exceeded the estimated normal cardiac weight by 313 gm. The T-waves were inverted in Lead I or in Leads I and II in six of eight cases (75 per cent). In two cases inversion of the T-waves was found in all leads but the interpretation of these changes was made somewhat difficult because of the administration of digitalis. In the group as a whole, but particularly in the cases uncomplicated by treatment with digitalis, there was a clear-cut tendency to inversion of the T-waves in Lead I or in Leads I and II.

Aortic Endocarditis With Stenosis or Insufficiency.—There were seven cases of aortic endocarditis with stenosis, or insufficiency, or both. The average age of the patients was fifty-five and five-tenths years (Table VI). The average blood pressure was 115 mm. systolic and 72 mm. diastolic. The average cardiac weight was 569 gm., which exceeded the average calculated normal weight by 290 gm. The T-wave was inverted in Lead I or in Leads I and II in five cases (71.4 per cent). There was inversion of the T-wave in all leads in two cases.

In this group, and in the previous group, in both of which there were aortic lesions, eleven (73.3 per cent) of the fifteen patients had inversion of the T-wave in Lead I or in Leads I and II and four in Leads I, II, and III (26.4 per cent).

Aortic Endocarditis With Mitral Stenosis.—In sharp contrast to this group of fifteen patients with aortic lesions, there was a group in which mitral stenosis complicated aortic stenosis or aortic insufficiency (Table VII). There were seven patients in this group, in six of whom the T-waves were inverted in Leads II and III (85.7 per cent). Two of these six patients had received digitalis, in only one of whom did the amount seem adequate to modify the T-waves. The average age of the patients in this group was fifty years. The average blood pressure was 126 mm. systolic and 68 mm. diastolic. The average cardiac weight was 498 gm., which exceeded the average calculated normal cardiac weight by 199 gm.

In combined aortic and mitral endocarditis there is the possibility of overload predominantly of the right or the left ventricle, depending in some measure on the degree of interference with function of the respective valves involved. Willius[30] has shown that the expectancy of life of patients with aortic endocarditis exceeds that of patients with mitral endocarditis by thirteen years. This indicates either that the strain on the heart, and in particular on the right ventricle, is greater from mitral endocarditis than is that on the left ventricle in aortic endocarditis, or that the right ventricle is less able to bear the strain than is the left ventricle. The occurrence of inversion of the T-wave in Leads II and III in patients in whom both aortic and mitral endocarditis are present may be correlated, possibly, with strain predominantly on the right side of the heart.

There was but one case of combined mitral and aortic endocarditis without definite evidence of stenosis or insufficiency of either valve (Table VII). There was perforation of the mitral valve, which may have produced some degree of mitral insufficiency. The T-waves were inverted in all leads in this case. An indeterminate amount of digitalis had been administered in this case before the electrocardiogram was taken.

Hypertension and Mitral Endocarditis.—When hypertension is complicated by mitral stenosis, again a group of cases is found in which the

TABLE IV

CASES OF PROBABLE PREEXISTENT HYPERTENSION WITH DEFINITE CORONARY SCLEROSIS

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
54	52 M	140	80	+	Left	600	142	Aortic valve stiffened; calcification of mitral ring	Negative	2+	2+		No digitalis given; markedly thickened left ventricle; T-wave upright in later electrocardiogram; no infarction
62	69 M	113	74	+	Left	500	249	Extreme calcification of aortic valve cusps, thick cusps at bases of mitral valve	Negative	4+	4+		No digitalis given; angina pectoris clinically; slight diffuse fibrous streaking scattered throughout the left ventricle
63	81 F			+	Left	450	187	Negative	Fine adhesions easily broken almost completely obliterating pericardial cavity	4+	4+	Cerebral hemorrhage; arteriosclerotic scarring of the kidney	No digitalis given; paroxysmal dyspnea; occasional fine fibrous streaking of myocardium

TABLE IV—CONT'D

73	55 F	160	100	+	Left	335		Negative	Negative	3+	3+		Four c.c. digitalis before electrocardiogram; no infarction; dilatation of left ventricle; exophthalmic goiter
87	62 M	164	90		Left	627	373	Negative	Several fibrous-like adhesions	3+	3+	Enlarged kidneys	Sixty minims digitalis before first and 800 before second and third electrocardiograms; first electrocardiogram normal, second showed inverted T-wave in Leads I-II-III, third diphasic T-wave in Leads I-II inverted in Lead III
109	50 M	139	88	+	None and right	357	333	Atheromatous changes in all valves grade 2+	Negative	3+	3+	Negative cerebral vessels; arteriosclerotic changes in the kidney; marked arteriovenous compression of the fundus oculi	No digitalis given

TABLE V

CASES OF SYPHILITIC AORTIC INSUFFICIENCY

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		FUNDUS OCULI	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
7	42 F	142	68	+	Left	340		Sclerosis 3+ of aortic	Negative	2+	2+		No digitalis given; hypertrophy of left ventricle 3+; angina due to almost complete occlusion of coronaries at orifices; inverted T-wave in Leads I-II-III in electrocardiogram taken during angina
24	43 F	115	30	+	Left	Heart dilated		Marked sclerosis of aortic and mitral	Petechial hemorrhages on visceral pericardium			Pupils	No digitalis given; some rheumatic history but case considered to be syphilitic aortitis; acute dilatation of heart; Argyll Robertson pupil
37	42 M	132	60	+	Left			Anterior aortic cusp scarred and adherent	Negative	0	0		No digitalis given; hypertrophy and dilatation of left ventricle; patient had angina with hemorrhagic infarction at apex of left ventricle; extensive calcification in origin of aorta

TABLE V—CONT'D

41	35 F	142	0	+	Left			Chronic syphilitic aortitis	Negative	2+	2+	Negative	No digitalis given; dilatation of left ventricle
59	62 M	154	41	+	None	550	341	Aortic ring dilated 3+; terminal vegetations	Negative	0	0		No digitalis given; diffuse cardiac hypertrophy and dilatation
89	52 M	120	84		Left	750	235	Aortic endocarditis with insufficiency (probably syphilitic)	Fibrous adhesions to pericardium and lung	2+	2+		Three c.c. digitalis before first electrocardiogram, and nine c.c. before second; first showed diphasic T-wave in Leads I-II and inverted T-wave in Lead III; second electrocardiogram showed inverted T-wave in Leads I-II-III; angina present, marked aortitis; left ventricle more hypertrophied and dilated than right
112	45 M	160	25		None	700	353	Retraction of aortic leaflets	Negative	3+	3+	Negative	Fourteen c.c. digitalis before second electrocardiogram; first electrocardiogram showed diphasic T-wave in Lead I; second showed inverted T-wave in Leads I-II-III; saccular aneurysm of ascending aorta 9 by 11 cm.; hypertrophy and dilatation of left ventricle
153	32 F	138	56	+	Left	760	300	Aortic insufficiency	Negative	2+	2+	Negative	Digitalis given 3 weeks before coming to the clinic; 6 c.c. digitalis given before first electrocardiogram; aortitis; dilatation of heart 4+

TABLE VI

CASES OF AORTIC STENOSIS AND INSUFFICIENCY

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		RENAL VESSELS	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
6	46 M	116	66	+	Left	656		Aortic stenosis 4+; small vegetations on mitral	Multiple small subepicardial hemorrhages	0	0		Two drams digitalis three times daily for one month previously; fibrous and fatty changes in heart; aortic valve completely closed
29	40 F	98	77	+	Left	675	215	Marked aortic stenosis	Negative	0	0		Inverted T-wave in Lead I of first electrocardiogram became diphasic on digitalis; second showed diphasic T in Lead I; hypertrophy and dilatation of left ventricle
35	62 M	122	60	+	Left	5 times normal size		Mitral admits five fingers; aortic stenosis and insufficiency; tricuspid admits seven fingers	Negative	0	0	Arteriosclerotic kidney	Right auricle markedly dilated and thinned; left ventricle hypertrophied 4+
82	62 M	105	89		None	646	306	Sclerosis and calcification 4+ of aortic valve	Negative			Arteriosclerotic changes	No digitalis given; inverted T-wave in Leads I-II-III; both ventricles hypertrophied
129	50 M	150	70		None	505	251	Aortic stenosis with calcification	Negative	0	0		No digitalis given; inverted T-wave in Leads I-II-III; hypertrophied left ventricle
142	54 M	100	75	+	None	504	235	Chronic aortic endocarditis with stenosis; slight mitral endocarditis with stenosis	Negative	2+			No digitalis given; greatly hypertrophied left ventricle; right coronary artery enlarged and tortuous
170	74 M	119	70	+	Left	428	388	Definite calcified aortic stenosis; sclerosis of mitral 1+	Negative	2+	2+		No digitalis given; angina clinically

								sclerosis of mitral 1+				really

TABLE VII

CASES OF AORTIC INSUFFICIENCY OR STENOSIS AND MITRAL STENOSIS

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS II-III	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT	
12	39 F			+	None			Marked stenosis of mitral and aortic	Negative	0	0	No digitalis given; marked aortic sclerosis; clinical diagnosis, mitral stenosis
70	53 M	76	46	+	Left	520	329	Marked aortic stenosis; slight tricuspid stenosis; sclerosis of aortic, mitral, and tricuspid; mitral stenosis	Negative	3+	3+	No digitalis given; first electrocardiogram showed diphasic T-wave in Lead I; marked dilatation of left ventricle; arteriosclerotic changes in renal vessels; negative fundus
83	62 M	180	80	+	Right	381	353	Aortic and mitral endocarditis	No adhesions	1+	1+	No digitalis given; exophthalmic goiter probably accounts for blood pressure; diagnosis, mitral stenosis and aortic endocarditis; hypertrophy and dilatation of both ventricles
99	58 M	120	78	+	Right	862	343	Fish-mouth aortic valve stenosis; partial contraction of mitral	Negative			Seven c.c. digitalis before electrocardiogram; generalized cardiac hypertrophy and dilatation
105	36 F	160	70	+	Right	452	225	Mitral stenosis 4+; aortic endocarditis 2+	Negative	0	0	Much digitalis before coming to the clinic; nauseated coupled beats; left auricle tremendously dilated; dilatation 2+ of left ventricle and 1+ of right
127	49 F	100	68	+	Left	250	272	Stenosis of aortic; some mitral stenosis; vegetations on tricuspid	Fibrous adhesions between two layers			No digitalis given; coronary sclerosis at orifice of left coronary
171	55 F	120	70		None	525	275	Aortic and mitral stenosis	Negative	1+	1+	No digitalis given; hypertrophy and dilatation of both ventricles; left auricle dilated 3+; inverted T-wave in Leads I-II
113*	26 M	134	60		Left	478	363	Bacterial endocarditis of aortic and mitral valves, perforation of mitral	Negative	0	0	Electrocardiogram taken before digitalis; dilatation and hypertrophy of both ventricles: inverted T in Leads I-II-III

*A case of aortic and mitral endocarditis without stenosis or insufficiency.

strain may be predominantly in the left or in the right ventricle (Table VIII). In one case the electrocardiographic changes were not constant and that fact together with much treatment with digitalis makes impossible an analysis of the changes in the T-wave. In a second case, mitral stenosis complicated systolic hypertension and a slight degree of aortic stenosis; the T-waves were inverted in Leads II and III.

In two cases in which hypertension was complicated by mitral endocarditis, without satisfactory evidence of stenosis or insufficiency, the T-waves were inverted in Lead I or in Leads I and II (Table VIII). In one of these a previous tracing, after administration of digitalis, showed inversions of the T-wave in Leads II and III; this phenomenon changed to inversion of the T-waves in Leads I and II, two and four weeks respectively, after treatment with digitalis had been discontinued.

Cases of Strain Exerted on the Right Side of the Heart.—To obtain cases of uncomplicated lesions which throw definite strain on the right ventricle in which the electrocardiograms show significant inversions of the T-wave is more difficult. First of all, few cases of mitral endocarditis produce significant changes in the T-wave. Mitral endocarditis is notoriously unlikely to exist as an isolated lesion. Administration of digitalis is so universal and thorough in this group that to obtain cases in which its influence can be excluded is difficult. There are certain pulmonic lesions, also, that seem adequate to throw strain predominantly on the right ventricle.

Three cases of mitral stenosis and of mitral stenosis and insufficiency were studied (Table IX). In one of these, in which digitalis was not given, the T-waves were inverted in Leads II and III. In the second case of inverted T-waves in Leads II and III, 15 c.c. of the tincture of digitalis had been administered before the tracing was taken, so that it is impossible to exclude from the interpretation the effect of digitalis. In the third case much treatment with digitalis, both before the patient came to the clinic and while under treatment here makes analysis impossible. In a case of marked mitral insufficiency, uncomplicated by administration of digitalis, inversion in Leads II and III was seen. In two cases of pulmonary disease producing an increased load on the right ventricle, there were inversions of the T-wave in Leads II and III (Table IX). In no instance were the T-waves inverted in Lead I or in Leads I and II. This result is further strengthened by the consideration already given of groups of cases of aortic endocarditis, with and without complicating mitral stenosis. The presence of mitral stenosis is attended by the frequent inversion of the T-wave in Leads II and III; this is in sharp contrast with the changes in the T-wave seen in aortic stenosis alone. In these groups, although they are small, there is a marked tendency to inversion of the T-wave in Leads II and III, which is not in accordance with the electrocardiographic features ob-

TABLE VIII

CASES OF HYPERTENSION AND MITRAL STENOSIS

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT RENAL AND RETINAL DATA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC		AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
88	69 M	220	80	None	471		Aortic cusps adherent for 6 mm.; mitral endocarditis with slight stenosis	Negative	1+	1+	Arteriosclerotic atrophy of the kidney; arteriosclerotic retinitis	T-wave inverted in Leads II-III
95	58 M	138	93	Changes from left to right in last four electrocardiograms	707	350	Marked calcification and puckering of mitral with considerable stenosis; slight aortic calcification	Normal	2+	2+		First electrocardiogram showed diphasic T-wave in Lead I; T-wave inverted in Leads I or I-II of second electrocardiogram; in Leads II-III; in Leads I-II-III of third, fourth, fifth and sixth electrocardiograms; digitalis before first, third, and fourth electrocardiograms; probably a hypertension, dilatation, and hypertrophy of heart; especially right auricle and ventricle
Cases of Hypertension and Mitral Endocarditis Without Stenosis or Insufficiency												
93	49 F	240	130	Left	810		Moderate thickening and vegetations of mitral and tricuspid	Negative	1+	1+	Renal blood vessels markedly thickened	No digitalis given; no clinical evidence of mitral or tricuspid disease; hypertrophy of both ventricles; inverted T-wave in Lead I
118	48 F	160	120	None	375	251	Old mitral endocarditis (no stenosis)	Negative	2+	2+		Digitalis before first electrocardiogram; inverted T-wave in Leads I or I-II of second and third electrocardiograms; in Leads II-III of first electrocardiogram; left ventricle wall thickened

TABLE IX

CASES OF RIGHT HEART STRAIN

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS II-III	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT	
						A. Cases of Mitral Stenosis						
155	51 M	130	100	+	Right	472	313	Thickening and vegetation of mitral stenosis	Negative	1+	1+	Digitalis tablets taken three times daily for ten days (elsewhere); we gave 18 c.c. before second electrocardiogram; on digitalis more or less for one year; left auricle dilated 4+ and right 2+; both ventricles 2+; diphasic T-waves in Leads II and III of first electrocardiogram, in Leads I, II and III of second electrocardiogram; right ventricle thicker than left.
161	34 M	120	96	(2) +	Right	429	300	Mitral stenosis 3+	Negative	0	0	No digitalis given; diphasic T-wave in Lead II of first electrocardiogram; inverted T in Lead III; thickened wall of right ventricle; negative fundi
164	33 M	135	104	+	None	707	300	Marked mitral stenosis and regurgitation	Negative	1+	1+	Fifteen c.c. digitalis before electrocardiogram; left auricle dilated 3+; hypertrophy of left ventricle; dilatation 3+ of both ventricles
						B. Case of Mitral Insufficiency						
180	45 F	155	80	+	None	385	342	Mitral thickened with marked insufficiency	Negative	1+	1+	No digitalis given; iso-electric T-wave in Lead I in first electrocardiogram
						C. Cases of Chronic Pulmonary Disease						
36	64 M	95	85	+	Right			Some sclerosis of anterior leaflet of mitral valve	Negative	2+	2+	No digitalis given; fibrosis of lungs; bilateral pulmonary tuberculosis; increased dyspnea and cough for four years; clubbed fingers five years
133	35 M	122	78	+	Right	470		Slight mitral endocarditis, no stenosis	Adhesions, parietal and visceral	1+	1+	No digitalis given; marked dilatation and hypertrophy of right ventricle; thrombosis of both pulmonary arteries (chronic)

served in conditions producing strain predominantly on the left ventricle; this demands study of a larger group of similar cases uncomplicated by treatment with digitalis.

Coronary Sclerosis.—We have discussed earlier in this paper, eight cases of definite coronary sclerosis associated with definite hypertension, and six cases associated with probable hypertension, in which the T-waves were inverted in Lead I or in Leads I and II in 86.6 per cent of cases. Later, thirteen cases with changes in the T-wave, associated with coronary sclerosis and myocardial infarction, will be discussed. We have only two cases of uncomplicated coronary sclerosis with significant changes in the T-waves occurring independently of hypertension or myocardial infarction. This strongly suggests that inversion of the T-wave in coronary disease is seldom seen except when an additional factor, such as hypertension or myocardial infarction, is present. Willius[27] has called attention to the fact that in 66 per cent of his series of cases of angina pectoris there were no significant inversions of the T-wave. We, likewise, in selecting the present cases from our necrospy material had to reject a number of cases of marked coronary sclerosis because significant changes in the T-wave had not been present. In the two cases of uncomplicated coronary sclerosis included in our series the average age of the patients was sixty-two and five-tenths years. The average blood pressure was 128 mm. systolic and 65 mm. diastolic. The average cardiac weight was 356 gm., which exceeded the average normal cardiac weight by 47 gm. In one case the T-waves were inverted in Lead I in association with incomplete bundle-branch block, and in one case the T-waves were inverted in all leads. Digitalis was not a factor in these cases.

Myocardial Infarction With Hypertension.—Twenty-one patients had myocardial infarction associated with definite hypertension (Table XI). Their average age was fifty-four and four-tenths years. The average blood pressure was 170 mm. systolic and 114 mm. diastolic. The average cardiac weight was 579 gm., which exceeded the average calculated weight by 252 gm. In fifteen cases (71.4 per cent) the T-wave was inverted in Lead I or in Leads I and II. In one case the T-waves were inverted in Leads II and III and were diphasic in Lead I. In two cases the T-waves were inverted in Leads II and III. In each of these cases the infarction was found in the posterior surface of the left ventricle. In two cases in both of which there was infarction in the posterior surface of the left ventricle the T-waves were inverted in all leads. There was one case of infarction in the left ventricle, in which the T-wave was inverted in Lead III only. There is little doubt that infarction takes precedence over strain predominantly of one ventricle in determining the type of changes in the T-wave that will be produced.

TABLE X

CASES OF CORONARY SCLEROSIS WITHOUT DEMONSTRABLE INFARCTION

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS			MISCELLANEOUS
		SYSTOLIC	DIASTOLIC		AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT	ANGINA	
160	66 M	140	70	None	340	304	Negative	Negative	3+	3+		No digitalis given, no infarctions; auricular fibrillation; inverted T-wave in Leads I-II-III
174	59 M	116	60	Left	373	294	Negative	Negative	2+	3+	+	No digitalis given; inverted T-wave in Lead I; incomplete bundle-branch block; lumen of anterior descending artery almost occluded; dilatation of left ventricle

Myocardial Infarction With Probable Preexistent Hypertension.—Eleven patients were studied in whom myocardial infarction complicated probable preexisting hypertension (Table XII). Their average age was sixty-two and seven-tenths years. The average blood pressure was 136 mm. systolic and 84 mm. diastolic. The average cardiac weight was 537 gm., which exceeded the average estimated cardiac weight by 209 gm. In seven cases there was inversion of the T-waves in Lead I or in Leads I and II in the electrocardiograms (63.6 per cent). In one case of inversions of the T-wave in all leads, infarction was present in both the anterior and in the posterior portions of the left ventricle. In one case an electrocardiogram showed a shifting type of change in the T-wave; there was infarction of the heart in both the anterior and the posterior portions. In one case inversion of the T-waves in Leads II and III was associated with infarction in the posterior region of the left ventricle. In another case inverted T-waves in Leads II and III were associated with massive, acute infarction in the posterior surface of the left ventricle. Intermittent incomplete bundle-branch block also developed in this case during the patient's illness, and he had a small infarct in the anterior portion of the interventricular septum, in the region supplied by the anterior descending coronary artery.

Myocardial Infarction With Coronary Sclerosis.—Thirteen cases of myocardial infarction associated with coronary sclerosis alone were studied (Table XIII). The average age in this group was sixty-two and six-tenths years. The average blood pressure was 131 mm. systolic and 81 mm. diastolic. The average cardiac weight was 400 gm., which exceeded the average estimated cardiac weight by 58 gm. In seven patients the T-waves were inverted in Lead I or in Leads I and II (53.8 per cent). In five cases there was inversion of the T-waves in Leads II and III, and in each case there was infarction in the posterior portion of the left ventricle and septum, in the area supplied by the right coronary artery. In one case the T-waves were inverted in Leads II and III, and subsequently, in association with infarction in the anterior and the posterior surfaces of the left ventricle, in Leads I, II, and III. Valvular lesions productive of selective strain on either ventricle were not present in these cases. The changes in the T-wave seemed to depend on myocardial infarction. All the cases of myocardial infarction will be considered in a subsequent study of their relation to inversion of the T-wave.

Myocardial Infarction With Miscellaneous Cardiac Diseases.—Seven patients in whom myocardial infarction was associated with miscellaneous cardiac diseases form this group (Table XIV). The average age of the patients was fifty-five years. The average blood pressure was 140 mm. systolic and 93 mm. diastolic. The average cardiac weight was 698 gm., which exceeded the estimated normal weight by 353 gm. In four cases there were inversions of the T-wave in Lead I or in

TABLE XI

CASES OF MYOCARDIAL INFARCTION ASSOCIATED WITH DEFINITE HYPERTENSION

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	ANGINA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT			
9	61 M	164	108		Slight right	626	300	Negative	Negative	1+	1+			Digitalis elsewhere; 3 c.c. digitalis at clinic before electrocardiogram; old and recent infarction in posterior portions of right and left ventricles and of interventricular septum; inverted T-wave in Leads II-III
16	58 M	178	118	+		478		Fusion of commissures at base of aortic leaflets	Negative	2+	3+	More than one-half of glomeruli hyalinized; retinitis of marked hypertension		No digitalis 17 days before electrocardiogram; chronic infarction of anterior portion left ventricle near apex and near base, also at obtuse margin of left ventricle
27	56 M	200		+	Left	525		Negative	Negative	4+	4+			No digitalis given; angina pectoris clinically
38	45 M	150	110	+	Left	700	363	Negative	Negative	3+	3+	Fundus oculi negative	+	No digitalis given; occlusion of anterior descending artery with ancient infarction
42	57 M	152	88	+	Left	650	353	Negative	Negative	2+	2+		+	No digitalis given; chronic infarction involving anterior surface of left ventricle, septum, and apex
52	52 F	170	135		None	480	248	Chronic mitral endocarditis; no stenosis or insufficiency	Negative	2+	2+	Marked renal arteriosclerosis		Eighteen and five-tenths c.c. digitalis before electrocardiogram; old infarction in posterior and anterior portion of left ventricle, more extensive in latter area; inverted T-wave in Leads II-III

TABLE XI—CONT'D

69	62 M	150	120	+	Left	575	343	Negative	Negative	3+	3+	Renal vessel walls thickened; retinal arteriosclerosis of hypertensive type		No digitalis given; infarction of lower anterior aspect of left ventricle and lower portion of posterior part of interventricular septum
86	59 M	140	120		None	425	333	Negative	Negative	3+	3+	Many renal glomeruli obliterated; small granular kidney; fundus oculi negative	+	No digitalis given; chronic myocardial infarction anterior surface of left ventricle; diphasic T-wave in Lead I; inverted T-wave in Leads II-III
92	54 M	240	130		Left	690	382	Negative	Negative	3+	3+			Six c.c. digitalis before electrocardiogram; chronic infarction of left ventricle; mural thrombus near apex; diphasic T-wave in Leads I-II (depression of S-T interval)
94	54 F	155	109		Right	496	282	Negative	Few fine adhesions parietal pericardium; dense adhesions to posterior surface of left ventricle	4+	3+	Moderate retinal arteriosclerosis of primary type	+	No digitalis given before electrocardiogram; infarction of posterior surface of left ventricle; complete occlusion of right coronary; inverted T-wave in Lead III only
107	54 M	160	140	+	None	421		Negative	Negative	2+	2+	Died of cerebral embolism; mild hypertension; arteriosclerosis of the fundus	+	No digitalis given; inverted T-wave in Lead I of second and third electrocardiograms; inverted T wave in Leads I-II-III in first electrocardiogram; acute and chronic infarction of anterior surface of left ventricle

TABLE XI—CONT'D

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	ANGINA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT			
108	58 M	150	100	+	Right	354	304	Negative	Negative	4+	4+	Renal vessel walls greatly thickened	+	No digitalis given; thrombosis of both coronary arteries; electrocardiogram normal before occlusion
115	54 M	158	112	+	Left	700	328	Negative	Negative	2+	3+	Slight sclerosis of renal vessels	+	No digitalis given; occlusion of left coronary artery with myocardial fibrosis of anterior two-thirds of left ventricle; coronary T-wave in Leads I-II
119	55 M	150	104		Left	846	353	Negative	Negative	1+	1+			No digitalis given; T-wave inverted in Leads I-II-III; chronic infarction in posterior surface of left ventricle
120	54 F	196	92	+	Left	475	315	Negative	Fibrinous exudate	1+	1+			No digitalis given; no infarction; thrombus in anterior descending branch of left coronary artery; left ventricle greatly thickened
137	50 M	220	140		None	870	300	Negative	Negative	1+	1+	Sclerosis 2+ of retinal arteries; retinitis of malignant hypertensive type		Much digitalis two months before coming to the clinic; inverted T-wave in Leads I-II-III; chronic infarction of posterior surface of left ventricle

TABLE XI—CONT'D

138	52 F	150	110	+	Left	567	394	Negative	Negative	1+	1+	Sclerosis 1+ of retinal arteries; retinitis of hypertensive type		No digitalis given; marked thickening of left coronary artery 3 cm. from orifice; chronic infarction of apex of left ventricle
141	46 M	170	130	+	Left	795	290	Negative	Negative	2+	2+	Renal vessel walls thickened		Fifteen c.c. digitalis before electrocardiogram; old infarction at apex and in anterior basal portion of left ventricle; recent infarction in posterior surface of left ventricle
151	38 F	190	120	+	Left	500	302	Negative	Fibrinous adhesions	1+	1+	Sclerosis 1, of retinal arteries with retinitis of benign hypertensive type		Electrocardiogram after 12 c.c. digitalis; acute infarction of anterior wall of left ventricle; wall of left ventricle thickened
159	60 F	185	94	+	None	463	276	Some sclerosis of mitral valves probably old healed endocarditis	Small patch old obliterative pericarditis 1.5 cm. in diameter	1+	1+	Stroke five months before; much arteriosclerotic thickening of the kidney		No digitalis given; infarction of left ventricle; antemortem thrombus of left auricle
181	64 M	142	100	+	Left	525		Sclerosis 3+ of mitral	Negative	3+	3+			No digitalis given; incomplete right bundle-branch block; left coronary artery occluded near origin with some infarction of anterior surface of left ventricle and interventricular septum

TABLE XII

CASES OF MYOCARDIAL INFARCTION ASSOCIATED WITH PROBABLE PREEXISTENT HYPERTENSION

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	ANGINA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT			
39	50 M	157	89	+	Right and left	552	325	Negative	Negative	4+ calcified	4+ calcified	Senile fibrosis of the fundus oculi	+	No digitalis given; also inverted T-wave in Leads II-III; infarction in left ventricle, both anterior and posterior surfaces, as well as infarction of posterior surface of right ventricle
57	58 M	121	81	+	Left	525	382	Negative	Negative	3+	3+	Renal arteriosclerotic changes; fundus oculi negative	+	No digitalis given; marked atrophy with some fibrosis in anterior and apical portions of the left ventricle
67	60 M	138	90	+	Left	598	490	Negative	Adhesion at apex	3+	3+			No digitalis given; chronic infarction at apex and interventricular septum; occlusion of anterior descending branch of left coronary artery
74	49 M	130	98	+	Left	715	343	Negative	Negative	2+	2+	Unconscious for twenty-four hours; renal arteriosclerosis 3+; reduction in caliber of arteries of the fundus		Eight c.c. digitalis before electrocardiogram; old infarction of left ventricle at obtuse margin extending from midventricle to within 2.5 cm. of apex and anteriorly to within 2.5 cm. of anterior interventricular sulcus; infarction midway between anterior and posterior interventricular sulci, all in distribution of right coronary artery; right bundle-branch block

TABLE XII—CONT'D

77	57 M	130	90		Left	520	353	Negative	No adhesions	3+	3+		+	No digitalis given; inverted T-wave in Leads I-II-III; old and recent infarctions of myocardium; old one on posterior surface of left ventricle, recent on anterior surface of left ventricle
106	58 M	112	64		None	555	300	Negative	Negative	3+	3		+	No digitalis given; inverted T-wave in Leads II-III; incomplete right bundle-branch block; extensive recent infarction posterior surface of left ventricle and septum; small region of recent infarction anterior surface of left ventricle and septum near apex
130	77 M	154	82	+	Right	425	313	Negative	Negative	3+	3+	Marked renal arteriosclerosis		No digitalis given; chronic infarction in anterior portions of left ventricle and septum
135	76 M	110	82	+	Left	582	294	Negative	Negative	3+	3+	Walls of renal vessels thickened		Two c.c. digitalis before electrocardiogram; complete right bundle-branch block; diffuse fibrosis throughout left ventricle in regions supplied by both coronary arteries
150	78 M	135	90	+	Left	318	223	Negative	Negative	3+,	3+	Renal arteriosclerotic atrophy; arteriosclerosis of the fundus oculi		No digitalis given; chronic infarction in anterior portion of the left ventricle and septum
162	64 F	158	86		Left	500	300	Negative	Practically negative	4+	3+	Much pitting of surface of kidney	+	No digitalis given; inverted T-wave in Leads II-III; ancient and recent infarction in area supplied by descending branch of right coronary artery involving posterior surface of left ventricle and right ventricle; abnormally placed interventricular septum
175	63 F	158	68	+	Left	562	288	Negative	Negative	1+	1+	Retinal arteriosclerosis		No digitalis given; chronic infarction of posterior surface of left ventricle

TABLE XIII

CASES OF MYOCARDIAL INFARCTION

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT RENAL AND RETINAL DATA	ANGINA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC			AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT			
2	54 M	146	86		Left	388	329	Negative	Negative	2+	3+			No digitalis given; diphasic T-wave in Lead I; sclerosis of pulmonary artery; infarction of anterior surface of left ventricle and apex
33	41 M	114	60		Right	368	363	Negative	Apex only adherent to heart	2+	4+	Renal vessel walls thickened	+	No digitalis given; infarction of posterior surface of left ventricle and apex; inverted T-wave in Leads II-III
43	56 M	120	90	+		400		Aortic cusps sclerosed 2+	Negative	3+	3+		+	No digitalis given; chronic infarction in anterior surface of left ventricle near apex
47	74 M	118	76	+	Left	404	414	Negative	Negative	2+	4+			No digitalis given; infarction of apex and lower anterior portion of left ventricle and septum
51	80 F	150	92		Left	431	272	Negative	Negative	3+	4+			No digitalis given; diphasic T-wave in Lead I; inverted T-wave in Leads II-III; thrombus 0.5 cm. upper one-third right coronary artery; infarction of posterior surface of left ventricle; electrocardiogram T_3 type
96	70 M	145	55	+	Left	496	384	Slight arteriosclerotic thickening	Negative	3+	3+	No definite renal arteriosclerosis		No digitalis given; chronic infarction (?); complete right bundle-branch block

TABLE XIII—CONT'D

104	72 M	128	82	+	None	373	255	Negative	Negative	4+	4+	Marked thickening of the renal vessel walls		Electrocardiogram taken after 11 c.c. digitalis; chronic infarction posterior wall of left ventricle in region supplied by left coronary artery; chronic pulmonary fibrosis (roentgen ray)
117	54 M	152	84		Left	355	392	Negative	Negative	1+	1+		+	No digitalis given; T-wave inverted in Leads II-III; coronary T-wave; old and recent infarction in posterior surfaces of left and right ventricles
125	69 M	100	80		None	295	323	Negative	Negative	2+	2+			No digitalis given; inverted T-wave in Leads II-III; chronic infarctions, extensive fibrosis and thinning of apex and posterior portion of left ventricle
149	57 M	110	75		Right	475		Negative	Fibrous adhesions at base of left ventricle	3+	4+	Arteriosclerotic scarring of the kidney; fundus oculi negative	+	No digitalis given; inverted T-wave in Leads II-III of first electrocardiogram; inverted T-waves in Leads I-II-III of second electrocardiogram; incomplete bundle-branch block; large chronic and recent infarction of anterior surface of left ventricle and chronic infarction of posterior surface of left ventricle; partial left hemiplegia
154	71 M	Not taken		+	Left	300		Negative	Negative	3+	3+	Sclerosis of renal arteries 1+	+	No digitalis given; fibrosis of myocardium; chronic infarction
163	56 M	140	90		None	480		Negative	Negative	4+	+		+	No digitalis given; inverted T-waves in Leads II-III; coronary T-waves; infarction of posterior portion of left ventricle and interventricular septum
179	60 M	148	98	+	Left	434	343	Atheroma of mitral 2+	Negative	3+	3+	Retinal vessels of small caliber	+	No digitalis given; infarction of lateral wall of left ventricle not extending to endocardium or epicardium; incomplete right bundle-branch block

TABLE XIV

CASES OF MYOCARDICAL INFARCTION ASSOCIATED WITH MISCELLANEOUS CARDIAC DISEASES

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		DISEASES	T-WAVE INVERSION LEADS I OR I-II	VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		RENAL VESSELS	ANGINA	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC				AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT			
8	67 M	110	68	Obliterative pericarditis; infarction	+	Right left	475	255	Negative	Completely obliterated	2+	3+	Negative	+	No digitalis given; inverted T-wave in Leads II-III; old infarction in anterior portion of left ventricle and apex; more recent infarction in posterior portion of left ventricle; cerebral vessels negative; fundus oculi negative
11	55 M	172	70	Hypertension; aortic stenosis; infarction		Left	543		Marked aortic stenosis	Negative	3+	3+			No digitalis given; chronic infarction of posterior one-third of left ventricle; inverted T-wave in Leads II-III; fundus oculi negative
17	66 M	140	100	Hypertension; infarction; obliterating pericarditis	+	Left	1000+	450	Negative	Obliterating adhesive pericarditis	3+	3+	Moderate sclerosis		No digitalis given; infarction of anterior surface of the left ventricle; definite hypertension

TABLE XIV—CONT'D

48	42 M	150	121	Syphilitic aortitis; infarction		Right	630	323	Thickened aortic valve along line of closure	Negative	2+	2+			Inverted T-wave in Leads II-III; 4.5 c.c. digitalis before electrocardiogram; syphilitic aortitis with insufficiency; old and recent infarction in the posterior portion of left ventricle; definite hypertension
49	29 F	100	80	Aortic stenosis; infarction			573		Marked aortic stenosis; mitral thickening without stenosis or insufficiency	Negative	1+	1+	Negative		Inverted T-wave in Leads I-II-III; no digitalis given; chronic diffuse fibrosis in anterior portion of left ventricle; left coronary almost completely occluded at orifice; sudden death
90	58 M	130	70	Infarction; obliterative pericarditis; mitral and aortic endocarditis	+	Left	960	353	Aortic leaflets shortened and thickened; some thickening of mitral cusps	Completely obliterated by adhesions	1+	1+			Digitalis for nine weeks before electrocardiogram (amount ?); chronic infarction of anterior portion of left ventricle anterior to the obtuse angle
116	68 M	210	120	Infarction; hypertension; hemopericardium	+				Negative	Hemopericardium	2+	3			No digitalis given; acute infarction with rupture of the anterior portion of the left ventricle

TABLE XV

MISCELLANEOUS DISEASES

CASE	AGE AND SEX	DISEASE	AVERAGE BLOOD PRESSURE		VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
			SYSTOLIC	DIASTOLIC		AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
15	67 M	Marked fibrosis and calcareous aortic ring; calcification of bundle of His with complete heart-block	171	70	Left			Marked fibrosis and calcification of aortic ring; moderate mitral fibrosis	Negative	2+	2+		No digitalis given; inverted T-wave in Leads II-III; right auricle especially dilated; marked sclerosis of aortic valves with calcification; clinical signs of mitral endocarditis with insufficiency; complete heart-block and bundle-branch block
30	68 F	Hypertension; chronic pulmonary disease	180	96	None	Moderate enlargement		Marked aortic valve sclerosis; moderate fibrosis mitral	Negative	2+	2+	Thickened and sclerosed renal vessels	No digitalis given; ten years cough with marked expectoration; bronchiectasis and purulent bronchiolitis; inverted T-wave in Leads II-III
40	37 M	Aneurysm; syphilitic aortitis	126	68	None	650 with aneurysm		Negative	Negative	0	0	Renal arteriosclerosis; fundus oculi negative	No digitalis given; marked torsion and fixation of aorta; no infarction; inverted T-wave in Lead I
44	76 M	Hypertension; aortic stenosis; coronary sclerosis	170	100	Left	775	313	Aortic markedly calcified; slight sclerosis of mitral valve; aortic stenosis	Negative	2+	3+	Walls of renal arteries greatly thickened; senile fundus	No digitalis given; inverted T in Lead I; enormous hypertrophy of left ventricle; left ventricle wall thickened

TABLE XV—CONT'D

61	48 M	Probable hypertension; pericardial adhesions	106	62	None	700		Negative	Chronic fibrous pericarditis; adhesions to anterior surface	1+	1+	Cerebral embolism; softening of right cerebrum	No digitalis before electrocardiogram; inverted T-wave in Leads I-II-III; alternating right and left bundle-branch block
75	46 F	Mitral stenosis; obliterative pericarditis	134	80	Right	425	198	Fish-mouth mitral stenosis	Completely obliterated	0	0	Cerebral embolus with left hemiplegia	Six c.c. of digitalis a week for two and five-tenths months before last electrocardiogram; inverted T-wave in Leads II-III in third electrocardiogram; first two electrocardiograms showed no T-wave changes; last electrocardiogram taken day of death and after digitalis
100	55 F	Aortic stenosis; chronic pulmonary disease	154	108	Right	384	255	Aortic endocarditis with stenosis 1+	Negative	1+	1+		No digitalis given; inverted T-wave in Leads II-III; healed tuberculosis; pleuritis and lymphadenitis; metastasis to lungs; purulent bronchitis; bronchopneumonia
122	64 M	Aortic stenosis and insufficiency; coronary occlusion	124	68	Left	820	410	Mitral valve cusps are thick; marked aortic stenosis and regurgitation	Negative	2+	2+		No digitalis before first electrocardiogram; 3 c.c. before second; inverted T-wave in Leads I-II; atresia left coronary orifice with occlusion; very little evidence of infarction
123	66 F	Hypertension; aortic stenosis	240	115	Right	509	297	Slight thickening of mitral; marked of aortic with two cusps joined	Few adhesions anteriorly	1+	1+	Moderate renal arteriosclerosis	No digitalis given; inverted T-wave in Lead I; markedly thickened left ventricle

TABLE XV—CONT'D

CASE	AGE AND SEX	DISEASE	AVERAGE BLOOD PRESSURE		VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		SIGNIFICANT CEREBRAL, RENAL, AND RETINAL DATA	MISCELLANEOUS
			SYSTOLIC	DIASTOLIC		AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
128	40 M	Obliterative pericarditis; mitral stenosis	150	65	Right	With pericardium 800	284	Fish-mouth stenosis of mitral; thickening of aortic; somewhat fused cusps	Adhesive pericarditis	1+	1+		No digitalis given; inverted T-wave in Leads II-III; hypertrophy and dilatation of left ventricle
136	58 F	Mitral stenosis; obliterative pericarditis	115	80	None	With pericardium and part of mediastinum 287	272	Fish-mouth mitral stenosis 3+; insufficiency 1+ with recent vegetations	Completely obliterated by fibrous adhesions	1+	1+	Multiple embolic infarctions with cerebral hemiplegia; renal arteriosclerotic changes 2+	Digitalis for two weeks before electrocardiogram; inverted T-wave in Leads I-II; auricular fibrillation
146	36 M	Tuberculous pericarditis with effusion	108	88	Left		353	Negative	Pericardium thickened, adherent over auricles and great vessels	0	0		No digitalis recorded; T-wave inverted in Leads II-III

TABLE XV—CONT'D

148	61 M	Aortic sclerosis; mitral endocarditis without stenosis or insufficiency	164	68	Left	313		Chronic mitral endocarditis with subacute exacerbation; no stenosis or insufficiency	Negative	2+	2+	Renal lesions negative	No digitalis given; inverted T-wave in Leads I-II-III; aortic sclerosis 4+ with marked dilatation of the aortic arch
152	68 F	Obliterative pericarditis; aortic insufficiency; mitral endocarditis	130	70	Right	550	250	Aortic valve irregularly puckered from sclerosis; mitral similarly affected; aortic insufficiency	Obliteration of pericardial cavity	1+	1+		Twelve c.c. digitalis before electrocardiogram; inverted T-wave in Leads II-III; polyserositis (Pick's disease)
156	56 M	Definite hypertension; obliterative pericarditis	180	110	Left	800	350	Negative	Fibrous pericarditis almost obliterating	2+	2+	Thickened renal vessels; retinitis of glomerulonephritis; reduced caliber retinal arteries	No digitalis given before electrocardiogram; inverted T-wave in Leads I-II
157	16 F	Mitral stenosis; obliterative pericarditis	108	58	None	505	180	Mitral stenosis	Obliterative fibrous pericarditis	0	0	Renal lesions negative	No digitalis given; inverted T-wave in Leads II-III

TABLE XVI

CASES OF ADHERENT PERICARDIUM

CASE	AGE AND SEX	AVERAGE BLOOD PRESSURE		VENTRICULAR PREPONDERANCE	WEIGHT OF HEART, GM.		VALVES	PERICARDIUM	CORONARY SCLEROSIS		CEREBRAL VESSELS	MISCELLANEOUS
		SYSTOLIC	DIASTOLIC		AT NECROPSY	ESTIMATED NORMAL			RIGHT	LEFT		
3	66 F	130	80	None	With pericardium, 495		Slight thickening of mitral valve; no stenosis	Cannot be separated from heart	1+	1+	Cerebral embolism; infarction in brain	No digitalis given; inverted T-wave in Leads I-II-III; auricular fibrillation; right auricle dilated 3+; no infarction
28	22 M	102	68	None			Moderate fibrous thickening of tricuspid, mitral, and aortic	Marked fibrous completely obliterative pericarditis	0	0		No digitalis given; inverted T-wave in Leads I-II-III; ascites; serofibrinous peritonitis; obliterative pleuritis and pericarditis (Pick's disease)
64	57 M	120	75	Left	675	353	Negative	Complete obliteration of pericardium	1+	1+		Six c.c. of digitalis before first electrocardiogram; fourteen c.c. before second; inverted T-wave in Leads I-II of second electrocardiogram; enormous hypertrophy of left ventricle
84	27 F	108	62	None			Negative	Heart fastened to base of pericardium by tumor mass	0	0		No digitalis given; inverted T-wave in Leads I-II; inferior surface of heart invaded by tumor mass
98	26 F	115	90	None	273	180	Negative	Complete obliteration of pericardium	0	0		No digitalis given; inverted T-wave in Leads II-III; small infarction in thickened pericardium on posterior surface of left ventricle but not involving myocardium

Leads I and II. In three cases the T-waves were inverted in Leads II and III. In two of the cases in the latter group conditions producing strain predominantly on the left ventricle were present, but the tendency of this to modify the type of changes in the T-wave was overbalanced by the effect of infarction in the posterior portion of the left ventricle. In one case in which the T-waves were inverted in all leads, chronic diffuse fibrosis in the anterior portion of the left ventricle existed.

Miscellaneous Cardiac Diseases.—A miscellaneous group of sixteen cases, difficult to classify because of multiple factors productive of myocardial strain, was studied (Table XV). A definite analysis of these cases in relation to strain exerted predominantly on one of the ventricles is not possible.

Chronic Adherent Pericarditis.—Five patients with chronic adherent pericarditis were studied (Table XVI). As pointed out before, it is impossible to say whether this condition exerts a strain predominantly on one or the other ventricle. The average age of the patients in this group was thirty-nine and six-tenths years. The average blood pressure was 110 mm. systolic and 75 mm. diastolic. The average cardiac weight was 481 gm., which exceeded the calculated average normal cardiac weight by 266 gm. In two cases there was inversion of the T-waves in Leads I, II, and III. In two cases the T-waves were inverted in Leads I and II, and in one of these sufficient digitalis had been administered before the tracing was obtained to lay its value open to question. In one case the T-waves were inverted in Leads II and III. Ventricular preponderance was present in only one case, a fact worthy of note although its meaning is not clear. These cases did not lend themselves to analysis on the basis of differential ventricular strain.

GENERAL COMMENT

From a consideration of 117 cases (Table XVII) of cardiac lesions which throw a definite or probable strain preponderantly on the left ventricle, it is seen that in 83.7 per cent of them there were inverted T-waves in Lead I or in Leads I and II in the electrocardiogram. In nine cases (7 per cent), there was inversion of the T-waves in Leads II and III. In seven of these cases there was myocardial infarction in the posterior portion of the left ventricle which accounted for the inversion of the T-waves in Leads II and III. In eleven cases (9.4 per cent), the T-waves were inverted in all leads. Certain of these cases may include those in which the inversion of the T-waves in Lead III is not of abnormal significance, and such cases would fall in the group with inversions of the T-wave in Leads I and II. Thus, when strain predominantly on the left ventricle produces inversion of the T-wave, the inversion is found in Lead I or in Leads I and II in a high percentage of cases.

In Table XVII six cases are recorded in which there were lesions which would throw strain predominantly on the right ventricle. In 83.3 per cent of these cases, the T-waves were inverted in Leads II and III and in no instance did there occur inversions of the T-wave in Lead I or in Leads I and II. In one case, the T-wave was diphasic in all leads. Here the association of inverted waves in Leads II and III with conditions which produce right ventricular strain is striking. The number of cases in this group is so small that in order to establish inversions of the T-wave in Leads II and III, as an expression of strain preponder-

TABLE XVII

DISEASES CAPABLE OF PRODUCING VENTRICULAR STRAIN

	DISEASE	CASES	HEART WEIGHT, GM.	EXCESS OF ESTIMATED NORMAL WEIGHT, GM.	LEFT VENTRICULAR STRAIN					
					PER CENT			CASES		
					INVERTED T IN LEADS I OR I-II	INVERTED T IN LEADS II-III	INVERTED T IN LEADS I-II-III	INVERTED T IN LEADS I OR I-II	INVERTED T IN LEADS II-III	INVERTED T IN LEADS I-II-III
1	Definite hypertension	42	604	280	90.5	4.7	4.7	38.0	2	2
2	Probable hypertension	13	607	258	100.0			13.0		
3	Definite hypertension and marked coronary sclerosis	9	584	252	88.8		11.2	8.0		1
4	Probable hypertension and marked coronary sclerosis	6	478	221	83.3		16.6	5.0		1
5	Syphilitic aortic insufficiency	8	620	313	75.0		25.0	6.0		2
6	Rheumatic aortic endocarditis	7	569	290	71.4		28.6	5.0		2
11	Myocardial infarction and definite hypertension	21	579	252	71.4	19.0	9.5	15.0	4	2
12	Myocardial infarction and probable hypertension	11	537	209	63.6	27.2	10.0	7.0	3	1
	Total	117			83.7	7.0	9.4	97.0	9	11
	Right Ventricular Strain									
9	Mitral stenosis	3	536	232		66.6	33.3		2	1
9	Mitral insufficiency	1	385	43		100.0			1	
9	Chronic pulmonary disease	2				100.0			2	
	Total	6				83.3	16.6		5	1
	Right and Left Ventricular Strain									
7	Mitral stenosis and aortic stenosis and insufficiency	7	498	199	14.3	85.7		1.0	6	
7	Aortic and mitral endocarditis	1	478	115			100.0			1
8	Hypertension and mitral stenosis	2	589	249		50.0	50.0		1	1
8	Hypertension and mitral endocarditis with stenosis	2	555	305	66.6	33.3		2.0		
	Total	12			22.8	62.0	15.2	3.0	7	2

antly of the right ventricle, the collection of further cases yielding the same result will be required.

There were twelve patients (Table XVII) with a combination of diseases, such that strain could be exerted on both ventricles. The strain on the right ventricle in all of these cases was produced by mitral endocarditis with or without stenosis. The T-waves were inverted in Leads II and III in seven of twelve cases (62 per cent). In three cases (22.8 per cent) the T-waves were inverted in Lead I or in Leads I and II. The incidence of inversion of the T-waves in Leads I and II (22.8 per cent as compared with 83.7 per cent) is much lower in diseases producing strain predominantly on the left ventricle when mitral endocarditis is associated than when it is absent. It has been suggested previously, in a discussion of the combined group of mitral stenosis, aortic stenosis and insufficiency, that when the two conditions compete in producing strain predominantly on the right or the left ventricle respectively, disease of the mitral valve may place a more serious strain on the right ventricle than that which aortic disease places on the left ventricle. If this assumption is correct, it is possible that a similar condition results at times in combinations of hypertension and mitral stenosis and that the predominant inversion of the T-waves in Leads II and III, shown in this group, is an expression of strain predominantly on the right ventricle, due to mitral valvular disease.

We have been unable to make any definite correlation between cardiac weight and the type of changes in the T-wave that occurred. For example, the average cardiac weight, and its excess over the calculated normal weight, was found to be essentially the same in definite or probable hypertension, associated with coronary sclerosis and without infarction, as that in the groups of mitral stenosis and mitral and aortic stenosis. Yet in the group with hypertension and coronary sclerosis the inversion of the T-waves is chiefly in Lead I or in Leads I and II and in the groups with mitral stenosis and with mitral and aortic stenosis, chiefly in Leads II and III. That large hearts give a high percentage of inversions of the T-wave in Lead I or in Leads I and II is explained by the fact that the conditions which produce the largest hearts are those conditions which produce a clear-cut strain on the left ventricle.

Attention has been directed to the fact that there were only two cases of coronary sclerosis unassociated with either definite or probable preexistent hypertension, or with myocardial infarction, in which significant inversions of the T-wave occurred. This seems to indicate that some additional factor, such as hypertension or myocardial infarction, is necessary in cases of coronary sclerosis to produce inversion of the T-wave. This seems a reasonable explanation of the fact, observed by

Willius[27] and others, that in many cases of angina pectoris there are no significant changes in the T-wave.

It must not be forgotten, furthermore, that inversions of the T-wave following infarction tend to disappear in from six months to two years, provided the patient survives.[19, 21, 24] Therefore, discovery of inversions of the T-waves in these cases will depend on the time the electrocardiogram is taken in relation to the time of infarction.

Fifty-two patients with myocardial infarction, either with or without hypertension, are included in this study. This number constitutes nearly 30 per cent of the total group and emphasizes the frequency with which myocardial infarction is found in patients who exhibit significant changes in the T-wave. The evidence at hand indicates that infarction is a more dominant factor than differential ventricular strain in determining the type of inversion of the T-wave produced. Further, infarction of the anterior portion of the left ventricle and septum (the region supplied by the left coronary artery) produces inversion of the T-wave in Lead I, or in Leads I and II, whereas infarction of the posterior portion of the left ventricle and septum (the region usually supplied by the right coronary artery) causes inversion of the T-wave in Leads II and III. These cases will be discussed in detail elsewhere.[2]

Numerous reports have appeared in the literature regarding the effect of administration of digitalis on the electrocardiogram. The recent observations of Bromer and Blumgart indicate that the earliest effect of digitalis is to change the amplitude of the T-wave in all leads. This phenomenon was observed by Cohn, Fraser, and Jamieson. Following this, Bromer and Blumgart observed that the R-T or S-T interval in Lead III became depressed, less elevated, or altered in general shape. The more pronounced effects which they obtained with greater dosage consisted of further alterations in the R-T or S-T interval, and they noted that the greatest change occurred in Lead III, slightly less in Lead II and least of all in Lead I. Pardee[20] has noted this peculiar modification of the S-T interval and has illustrated it by figures in his text. There is a distinct tendency exhibited in his tracings for the T-waves to become diphasic as a result of the modification of the S-T interval. Kerr, in discussing a paper by Berman and Mason, stated that he had noted development of an inverted T-wave in Lead III more frequently than in Leads I and II following treatment with digitalis. We have observed the peculiar depression of the R-T interval in our tracings following administration of digitalis and particularly in Leads II and III. At times, the T-waves are actually inverted in those leads. Obviously the effect of administration of digitalis on changes in the T-wave is highly important in the analysis of cases such as those presented in this study.

THEORETIC CONSIDERATION

The facts presented in this paper warrant considerable discussion in relation to the mechanism of production of inversion of the T-wave. The facts are oriented best when considered in the light of the evidence presented by Wilson and Herrmann that the T-wave of the normal ventricular complex is a combination of right and left ventricular effects. Lewis[15] interprets their study to mean that "In Lead III and usually in Lead II the end phase of the dextrocardiogram would be directed downward and in the levocardiogram it would be the reverse." In that case the upright T-wave of the normal electrocardiogram would be attributable to a preponderance of the right ventricular effect in Lead I and of left ventricular effects in Leads II and III. In a consideration of events in the formation of the T-wave in bundle-branch block, Wilson and Herrmann call attention to the fact that the upstroke of the T-wave in right bundle-branch block is due chiefly to the early decline of the process of excitation in the left ventricle. However, they admit that it may be due to preponderant right ventricular effects in the form of preponderance of retained activity on the right side of the heart. They stated that there is as much basis for the latter view as for the former.

It is necessary to consider conditions present in the individual fractionate components of cardiac muscle in normal or abnormal states to understand factors which determine changes in the T-wave. It seems justifiable to consider that ventricular strain results in a disturbed physiological status of the fiber, best described as a state of fatigue. The most important factor in fatigue in muscle has been shown to be the hydrogen-ion concentration.[1, 8, 11, 23] The conduction time and excitability of the cell are decreased by an increase in the hydrogen-ion concentration.[1, 8] Fulton stated that in fatigue some fibers are more affected than others (leading to asynchronism). Mines stated that a slight increase in hydrogen-ion concentration diminishes the duration of the electrical change in cardiac muscle. Redfield and Edsall studied the effect of fatigue in the ventricle of the tortoise, in an oxygen-free atmosphere, and found that the amplitude of contraction decreased logarithmically to the point of extinction, that the amount of lactic acid increased step by step with the degree of fatigue, and that the duration of contraction does not increase in cardiac muscle as it does in skeletal muscle when fatigued. The results summarized here indicate that fatigue or increased hydrogen-ion concentration diminishes amplitude of contraction, duration of electrical effect, conductivity, and excitability in cardiac muscle. It seems reasonable to expect these changes to be manifest predominantly in the ventricle that is subject to the greatest strain.

Katz and Weinman consider that the T-wave is the result of asynchronous cessation of electrical activity in the fractionate components of heart muscle. They consider that differences in initial tension and arterial resistance in the two ventricles and variable nutrition in different regions of the ventricles are factors capable of producing variation in the duration of fractionate contractions. If the conclusion of Wilson and Herrmann is accepted, that the T-wave is a combination of right and left ventricular effects, then three main possibilities may be considered as to the cause of inversion of the T-wave in Leads I and II in left ventricular strain, with its consequent disturbance of the physiological status of the muscle fiber. First, there may be a disturbance in electropotential balance, due to diminished duration of the electrical change in many or in all of the fractionate components of the left ventricle; second, the disturbance in electropotential balance may be due to early decline of the process of excitation in certain or in all of the fractionate components; third, the preponderance of retained activity in the relatively normal right ventricle may be the factor determining the inversion of the T-wave in these leads. In right ventricular strain, on the other hand, with inverted T-waves in Leads II and III, the first two of these three factors may be conceived as acting in the right rather than in the left ventricle; and the third factor may be exerted in the left rather than in the right ventricle. This consideration is based on the supposition that a plane exists in the heart about which the right and left ventricles act as opposing forces in their effect on the T-wave. That this plane is one accurately separating the right and left ventricles seems questionable on the basis of some observations we have made in infarction of the left ventricle.

We have called attention to our observation that infarction of the anterior portion of the left ventricle and apex is associated with inversion of the T-wave in Leads I and II, whereas infarction in the posterior surface of the left ventricle and posterior one-third of the septum in the region usually supplied by the right coronary artery produces inversion of the T-wave in Leads II and III. In other words, the infarction in the latter region produces the same changes in the T-wave as those encountered in strain of the right ventricle. This similarity in effect suggests that the posterior part of the left ventricle and septum may act with the right ventricle to produce an electrical change opposed in the direction of its action on the T-wave to that produced by the anterior two-thirds of the left ventricle and septum and that the resultant of these forces determines the direction of the T-wave. If this observation is valid, then the plane separating the electrical forces which exert an influence on the T-wave divides the left ventricle in such a way that the posterior part of the left ventricle and posterior one-third of the septum act with the right ventricle. This may be one reason why strain on the right ventricle infrequently produces inver-

sion of the T-wave; the muscular mass of the right side of the heart is relatively small in comparison with the anterior two-thirds of the left ventricle and septum. Wilson and Herrmann attempted to explain how the thin-walled right ventricle at times can produce effects of greater amplitude on the T-wave than the thick-walled left ventricle. This fact would be rendered much more comprehensible if it is true, as it appears to be, that a portion of the left ventricle acts in conjunction with the right ventricle in producing electrical effects relating to the form and direction of the T-wave.

The possibility must be borne in mind that preponderant hypertrophy of the left or of the right ventricle plays an important rôle in determining the changes in the T-wave seen in differential ventricular strain. It is true that preponderant hypertrophy of one verticle bears

TABLE XVIII

VENTRICULAR PREPONDERANCE IN THE CASES STUDIED

DISEASES	CASES	PREPONDERANCE WITH INVERTED T-WAVE IN LEADS I-II			PREPONDERANCE WITH INVERTED T-WAVE IN LEADS II-III			PREPONDERANCE WITH INVERTED T-WAVE IN LEADS I-II-III		
		RIGHT	LEFT	NO PREPONDERANCE	RIGHT	LEFT	NO PREPONDERANCE	RIGHT	LEFT	NO PREPONDERANCE
Definite hypertension	37	1	26	6	1	1	0	0	0	2
Probable hypertension	11	1	9	1	0	0	0	0	0	0
Definite hypertension and definite coronary sclerosis	9	0	7	0	0	0	0	0	1	1
Probable hypertension and definite coronary sclerosis	6	0	4	1	0	0	0	0	1	0
Syphilitic aortic insufficiency	8	0	5	1	0	0	0	0	1	1
Aortic stenosis and insufficiency	7	0	4	1	0	0	0	0	0	2
Aortic insufficiency and mitral stenosis	8	0	0	1	3	2	1	0	1	0
Hypertension and mitral stenosis	5	0	2	1	0	0	1	1	0	0
Strain on right side of heart	6	0	0	0	4	0	2	0	0	0
Coronary sclerosis	1	0	0	0	0	0	0	0	0	0
Myocardial infarction with definite hypertension	20	1	10	4	2	0	1	0	1	1
Myocardial infarction with probable hypertension	7	1	5	0	0	0	1	0	0	0
Myocardial infarction	11	0	4	2	1	2	2	0	0	0
Myocardial infarction with miscellaneous diseases	6	0	2	1	1	1	0	0	0	1
Miscellaneous diseases	14	1	3	2	4	1	2	0	1	0
Adherent pericardium	5	0	1	1	0	0	1	0	0	2
Total	161	5	82	22	16	7	11	1	6	11

a fairly constant relationship to strain predominantly of that ventricle. It is possible that this hypertrophy may modify the period of activation or the electrical potential available for an effect on the T-wave and thus disturb the normal electrical balance between the two ventricles.

The factors producing preponderance in the electrocardiogram can be correlated only roughly with the type of inversion of the T-wave (Table XVIII). In general, inversion of the T-waves in Lead I or in Leads I and II is attended by a high incidence of left ventricular preponderance, whereas there is a fairly high incidence of right ventricular preponderance when the T-waves are inverted in Leads II and III. There was no evidence of preponderant influence of one ventricle in the electrocardiogram in one-fifth of the patients who exhibited inversion of the T-wave in Lead I or in Leads I and II, and one-third of the patients in whom inversions of the T-wave occurred in Leads II and III gave no evidence of preponderance of one ventricle. In nearly two-thirds of the patients with simultaneous inversions of the T-wave in all leads of the electrocardiogram, there was no evidence of ventricular preponderance. It seems evident that the factors that determine ventricular preponderance are not the same as those responsible for types of inversions of the T-wave. We cannot deny the probability that there may be some factors in common in the two processes.

It is well recognized that patients die from strain predominantly of the right or of the left ventricle, without ever showing significant inversions of the T-wave in the electrocardiograms. The studies of Willius[27, 31] have shown that in cases in which the heart was subjected to strain predominantly on one side, and in which there were inversions of the T-wave, the prognosis was much worse than in cases in which there were comparable lesions but in which significant inversion of the T-wave did not occur. The question naturally arises as to what determines the time at which changes in the T-wave make their appearance in conditions of prolonged ventricular strain as observed, for example, in hypertension or in aortic insufficiency. The most logical answer to this seems to be that it is a question of the degree to which overwork and fatigue interfere with the normal physiological activity in the muscle cells. It must be true, as in other tissues in the body, that a wide range of physiological adjustment or compensation can take place in heart muscle which is subjected to fatigue or to overstrain. However, it is probable that when fatigue or strain reaches or exceeds a certain limit, uncompensated metabolic disturbances occur, capable of modifying the electrical forces produced by the right and left portions of the heart and thus to bring about significant inversions of the T-wave. If these assumptions are correct, a rational basis is furnished for the poor prognostic outlook of patients in whose electrocardiograms significant inversions of the T-wave occur.

Finally, it should be mentioned that Daly, in experiments on dogs, in which he was able to place varying work loads on the two ventricles, found that, when the left ventricle performs excessive work, increased positivity of the T-wave in Lead III, and increased inversion of the T-wave in Lead I resulted. In two of Daly's experiments, performed on animals in which the T-wave in Lead III was inverted, the pulmonary artery was partly occluded by a clamp. In one of these experiments, application of the clamp caused an increase in the amplitude of the inverted T-wave. These results are in accord with changes in the T-wave observed in this study in relation to strain predominantly of the right and of the left sides of the heart. Furthermore, Otto has observed that in axial leads in the dog sudden increased work imposed on the right ventricle produced temporary inversion of the T-wave, while a sudden increase in the work imposed on the left ventricle tended to produce the opposite effect on the T-wave.

DIAGNOSTIC INDICATION OF INVERSION OF THE T-WAVE

If our interpretations of the facts presented in this paper are correct, then an inverted T-wave in Lead I or in Leads I and II is indicative of a lesion throwing strain predominantly on the left ventricle, whereas an inverted T-wave in Leads II and III indicates a lesion throwing a strain predominantly on the right ventricle. If conditions recognized as throwing a definite strain on the left ventricle, such as aortic stenosis or hypertension, show an inverted T-wave in derivations II and III, then we are led to suspect that, in addition, some lesion is present causing overload of the right ventricle or that infarction has occurred in the posterior portion of the left ventricle.

SUMMARY AND CONCLUSIONS

1. One hundred seventy-seven cases in which there were significant changes in the T-wave, and in which necropsy was performed, have been studied for evidence of relation between differential ventricular strain and the type of changes in the T-wave.

2. In cases in which the strain was thrown predominantly on the left ventricle, there was inversion of the T-waves in Lead I or in Leads I and II (84 per cent) and in only 7 per cent were the T-waves inverted in Leads II and III. Infarction was found in the posterior surface of the left ventricle in all but two cases in which electrocardiograms showed inversion of the T-waves in Leads II and III.

3. In cases in which the condition produced disproportionate load on the right ventricle, T-waves were inverted in Leads II and III in 83 per cent and no case showed inversion of the T-wave in Lead I or in Leads I and II.

4. In conditions in which multiple lesions compete for maximal strain on the two ventricles, the leads in which the T-waves will be

inverted cannot be predicted. It seems likely that the type of inversion of the T-wave encountered may be related to the ventricle suffering the greater stress in accordance with the two previous observations.

5. This differential effect of right or left ventricular strain on the type of inversion of the T-wave is in partial accord with the conclusion of Wilson and Herrmann that the T-wave of the normal ventricular complex is a combination of right and left ventricluar effects. The inversions encountered here are considered to be due to interference with the normal summation of right and left ventricular effects, that in turn are due to a disturbance of physiological conditions and electrical effects in the ventricle subjected to excessive strain.

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THE ELECTROCARDIOGRAM IN PERICARDIAL EFFUSION*

I. Clinical

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FOUR years ago one of us (R. W. S.) observed a patient with the typical signs of acute pericardial effusion whose electrocardiogram exhibited the monophasic R-T deviation characteristic of recent myocardial infarction. The curves were indeed so typical that the clinical diagnosis of coronary thrombosis and myocardial infarction was submitted. Much to our surprise the post-mortem examination revealed no significant changes either in the myocardium or in the coronary arteries, but a hemorrhagic effusion into the pericardial sac from a ruptured aneurysm of the ascending aorta. More recently we have seen an additional case of acute pericardial effusion with similar, although not such marked, changes in the electrocardiogram. These clinical observations appeared of sufficient importance to merit further study of the question in experiments on animals. The details of the experimental observations are presented in the paper following this one, but it may be stated here that acute pericardial effusion in dogs produced changes in the electrocardiogram similar to those observed in man.

CASE REPORT

A colored male, G. H., 53 years old, a laborer, was admitted November 9, 1925, complaining of breathlessness and attacks of substernal pain. He had had a primary luetic infection 27 years previously, otherwise he had enjoyed good health. The patient stated that he had been able to do manual labor until the onset of his present trouble one month before. This had begun with paroxysmal attacks of substernal pain, radiating to the neck, and later breathlessness on exertion appeared. However, he had not been bedridden until two days before admission when he was seized with a severe substernal pain and great respiratory distress.

Physical Examination.—This revealed a well-developed, colored male in acute distress from orthopnea. The pupils were small, unequal, and reacted in accommodation but not to light. There was no visible precordial activity. The area of cardiac dullness extended to the mid-axillary line in the fifth and sixth intercostal spaces, and 2 cm. to the right of the sternum in the third and fourth intercostal spaces. The heart sounds were quite muffled, but no adventitious sounds were audible. Palpation of the larger accessible arteries showed a marked diminution in pulse volume with the typical pulsus paradoxus. The blood pressure was 110 mm. Hg. systolic and 80 mm. Hg. diastolic. The signs of compression were elicited over the left lung base posteriorly. The breath sounds were accentuated throughout, but there was no demonstrable moisture at the lung bases. The liver extended almost to the umbilicus in the midline but was not tender, and there

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was no edema over the sacrum or the lower extremities. The patellar and Achilles tendon reflexes were absent on both sides. The blood Wassermann was four plus. The spinal fluid showed an increased globulin content, 150 small mononuclear cells per cubic millimeter and a four plus Wassermann reaction. Fluoroscopically,

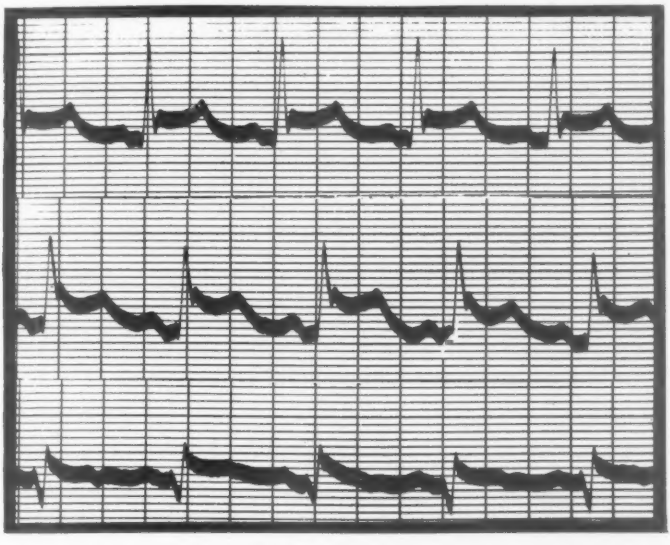

Fig. 1.—Three leads in a case of hemopericardium from a ruptured aneurysm. Note the abnormal ventricular complexes; the positive R-T deviation in Leads I and II, and the S-T segment above the iso-electric level in Lead III. In this figure and those following, the vertical lines represent 0.2 sec.; the horizontal lines 0.1 millivolt.

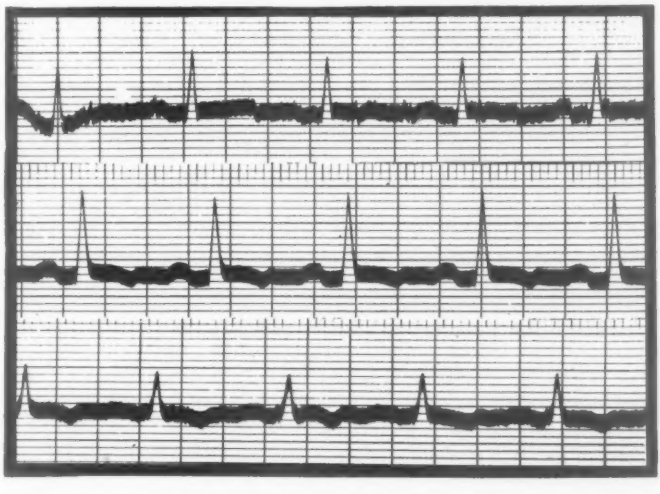

Fig. 2.—From the same patient thirty-three days after Fig. 1, and one week before death. Note the change in the ventricular complexes.

in the anteriorposterior view a marked increase was observed in the transverse diameter of the heart shadow to the right and to the left, and also an increase in the width of the great vessels. There was no appreciable movement of the heart with respiration, and the cardiac pulsations were very feeble. Projecting

into the lung field from the region of the right auricle there was a tumor mass the size of a billiard ball which did not pulsate. On rotating the patient this mass appeared to be posterior to the ascending aorta but did not infiltrate the lung field.

Clinical Course.—Since the clinical picture and fluoroscopic findings in this patient suggested pericardial effusion; the day following admission, a paracentesis of the pericardium was attempted, and 5 c.c. of blood were withdrawn, but it was thought at the time that this came from within the heart. A day later the electrocardiogram shown in Fig. 1 was obtained. This record was regarded as reliable evidence of recent myocardial infarction, and accordingly the patient throughout the remainder of his stay in the hospital was treated as a case of coronary thrombosis. He continued to have a low grade fever but never above 38° C. Dyspnea became more marked, the pulse volume diminished, edema of the lower extremities appeared, and the patient died November 20, 1925, forty-two days after admission. Seven days before his death the electrocardiogram shown in Fig. 2 was obtained. The following clinical diagnosis was submitted: Coronary arteriosclerosis with occlusion; myocardial infarction; cardiac hypertrophy and dilatation; syphilitic aortitis; aneurysm of the ascending aorta; cerebrospinal syphilis.

Autopsy Findings.—The heart, pericardium and aorta were removed *en masse*. On opening the pericardium 200 c.c. of hemorrhagic fluid escaped. Both the visceral and parietal layers were thickened, in some areas as much as 3 mm. The surfaces were markedly blood tinged, irregular and covered by a bloody, partially organized exudate. Throughout the pericardial cavity there were numerous bands of adhesions, some measuring 3 mm. in diameter. On cut section of both the visceral and parietal pericardium the deeper portions were edematous and showed marked vascularization. The heart was flabby and dilated but contained no antemortem thrombi. The mural endocardium and cardiac valves showed no evidence of disease. The columnae carneae and papillary muscles were enlarged and flattened, particularly on the right side. Several cut sections of the myocardium showed no gross areas of fibrosis or infarction. The coronary arteries were not narrowed at their mouths and when opened showed only moderate intimal changes. No thrombosis or reduction in lumen was found. The aorta was markedly dilated and inelastic throughout the ascending portion, the arch, and in the first few centimeters of the thoracic descending portion. The intima was thickened and corrugated and presented the typical gross appearance of syphilitic aortitis. Four centimeters proximal to the orifice of the innominate artery there was a circular opening in the aortic wall 3 cm. in diameter leading to a small aneurysmal sac 5 cm. in diameter, which was filled with a mottled, friable thrombus. On removing this thrombus one saw in the bottom of the sac a rupture 12 mm. in diameter which connected directly with a cavity in the visceral pericardium containing 175 c.c. of recent blood clot. The wall of this cavity contained an organized exudate which in some areas was elevated by recent hemorrhage.

Anatomical Diagnosis.—Syphilitic aortitis; saccular aneurysm of the ascending aorta with rupture into the pericardium; hemopericardium; organized pericarditis; chronic passive congestion of viscera; moderate hypertrophy and dilatation of the heart.

DISCUSSION

The clinical course in the above case may be interpreted in the light of the autopsy findings as follows: The long-standing syphilitic process in the aorta led to characteristic changes in the vessel wall with the formation of an aneurysmal sac opening 4 cm. proximal to the innomi-

nate artery. The process, however, spared the aortic ring, the valve leaflets, and the mouths of the coronary arteries, so that the heart was not embarrassed, and, as one frequently observes in such cases, the patient was able to work at manual labor until two months before death. His first symptoms—attacks of substernal pain and dyspnea—continued for three weeks, at which time he had an acute attack of substernal pain accompanied by great respiratory distress which incapacitated him and for which he sought admission to the hospital. It seems likely that this attack occurred at the time of rupture of the aneurysm into the pericardial sac; more certain is it that the clinical picture observed on admission was due to hemopericardium. As stated above, our initial impression of the case was pericardial effusion, hence an exploratory paracentesis was done, but not suspecting hemopericardium, the blood obtained from the puncture was thought to have come from the heart cavity. The day following, the electrocardiogram (Fig. 1) was obtained. This appeared to afford indisputable evidence of a recent cardiac infarct, and our original diagnosis of pericardial effusion was disregarded. The attack of substernal pain and dyspnea two days before admission was now ascribed to coronary thrombosis, and the clinical course of progressive circulatory failure was explained on the basis of myocardial infarction.

Referring now to the electrocardiogram, Fig. 1, it is clear that these curves display the positive S-T deviation seen in recent myocardial necrosis. They are typical with one exception; the S-T segment is above the iso-electric level in all leads, whereas in clinical curves the S-T segment is usually oppositely directed in Leads I and III, thus an S-T elevation in Lead I is accompanied by an S-T depression in Lead III and vice versa.

That characteristic abnormalities in the R-T segment of the electrocardiogram are associated with myocardial necrosis is a fact well established on both experimental[1, 2, 3, 4, 5, 6] and clinical[7, 8, 9, 10, 11, 12, 13, 14] grounds, and need not be discussed here; but so far as we are aware, there has appeared no evidence to show that effusion into the pericardial sac per se causes a deformity of the S-T segment similar to that seen in recent myocardial infarction. With the exception of coronary occlusion, and in moribund states, the only clinical curves showing similar R-T deviations are those obtained from patients with rheumatic carditis, and it is generally assumed that the deviation is due to the rheumatic myocardial lesion. In their paper on "Electrocardiographic Evidence of Myocardial Involvement in Rheumatic Fever," Cohn and Swift[13] present two curves from patients showing a positive R-T deviation, but one cannot ascertain from reading their paper whether or not these patients had a complicating pericardial effusion. Recently Porte and Pardee[14] reported three cases of rheumatic pericarditis with curves showing a slight upward convexity of the S-T segment preceding a

negative T-wave—the so-called coronary T-wave. The authors ascribed these changes to the rheumatic myocardial lesions and state: "We believe that the T-wave abnormality observed in these three cases of pericarditis is due to a complicating myocardial inflammatory reaction." Although Porte and Pardee[14] entitle their paper: "Coronary T-wave in Rheumatic Pericarditis," no significance is attached to the pericardial lesion or to the effusion in the pericardial sac in spite of the fact that the pathological report in their one autopsied case reads: "The pericardium contains an excess of hemorrhagic fluid."

Considerable difficulty is frequently encountered in determining the significance of myocardial lesions in electrocardiographic abnormalities. The above case of hemopericardium with no complicating myocardial lesion is a particularly good clinical illustration of the effect of hydrostatic pressure in the pericardial sac on R-T deformities in the electrocardiogram. We have observed other clinical cases in which the evidence was suggestive but not so conclusive, and we therefore hesitate to present them as clear-cut examples. Two, however, are sufficiently instructive to merit a brief discussion.

The first of these was a case of pyopericardium in a white male, aged 56 years, who was seen by one of us (H. S. F.) six days after the onset of an acute illness which began with a chill, fever, and later pain in the left chest. He had the physical signs of effusion over the left lower lobe, and the initial diagnosis was empyema complicating pneumonia. Pus was aspirated from the left chest. Seven days after admittance to the hospital, and thirteen days after the onset of this illness, the electrocardiogram reproduced in Fig. 3 was obtained. The positive S-T segment in Leads I and II suggested a coronary lesion with myocardial infarction. Eight days later, and twelve hours before death, the record in Fig. 4 was made, which displays a more normal appearance. At the post-mortem examination the pericardium contained 400 c.c. of a thick, viscid pus similar to that found in the left pleural cavity. No thrombi or narrowing of the coronary arteries were observed and the endocardium, the heart valves, and myocardium showed no gross changes. Histologically, sections of the myocardium showed obscure striations of the muscle fibers, which were larger than normal in some areas. Some fragmentation and segmentation of the muscle fibers was also observed. The epicardium was markedly thickened, measuring from 4 to 6 mm. in width. In the superficial layers there was an abundant fibrin deposit with a moderate number of round cells, mononuclears and polymorphonuclears. In the deeper layers there was organization of the exudate with numerous fibroblasts, and a moderate round cell infiltration. A section stained by Gram's method showed a few gram positive lancet-shaped diplococci (pneumococci) in the exudate.

The above case of purulent pericarditis with effusion affords suggestive but not conclusive evidence, since there remains the question of the possible effect of the myocardial damage associated with infection. At all events, this observation proves that the R-T deviation cannot be accepted as indisputable evidence of myocardial infarction, and further points to the value of such deviations in the early diagnosis of effusion in the pericardial sac.

Another case exhibiting R-T abnormalities was one of rheumatic carditis in a colored boy, 16 years old, who was seen two weeks after the onset of a typical attack of acute rheumatic fever with multiple migratory arthritis. When first observed, the patient was acutely ill with fever, dyspnea and orthopnea. The area of cardiac dullness was definitely increased both to the right and to the left

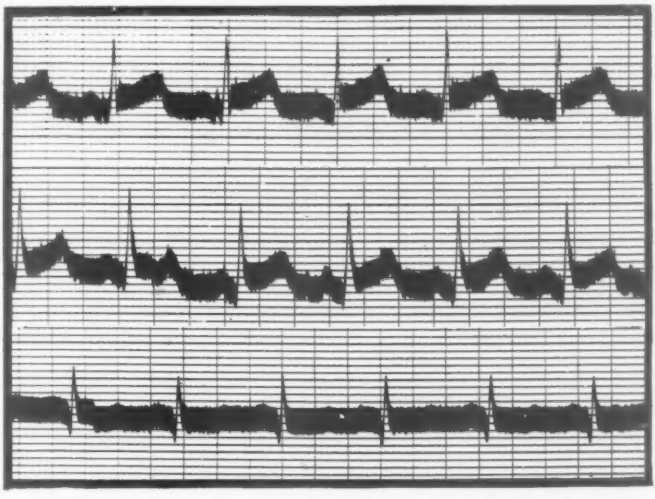

Fig. 3.—Three leads from a case of purulent pericarditis with effusion. Note the high take-off of the S-T segment in Leads I and II which merges with the T-wave.

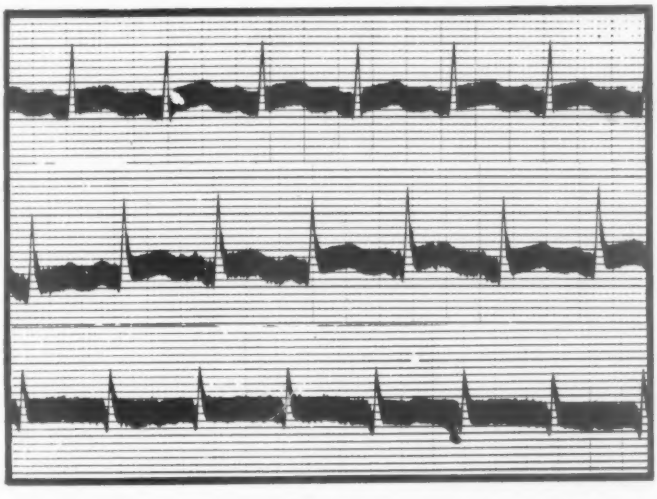

Fig. 4.—From the same patient eight days after Fig. 3, and twelve hours before death. Note the approach of the S-T segment and T-wave toward the iso-electric level.

(verified by x-ray), the heart sounds were muffled, signs of compression were elicited over the left base posteriorly, and a friction rub was audible over the precordium. At this time the record shown in Fig. 5 was made, and nineteen days later when the patient was much improved clinically, the record in Fig. 6 was obtained. Pericardial effusion was suspected in this case but never proved, hence we cannot

conclude that the R-T deviation in Fig. 5 was caused by pericardial effusion. However, since this possibility existed, the conclusion that the rheumatic myocardial lesion caused the R-T deviation is not acceptable without further proof.

We are not contending here that rheumatic myocardial lesions may not also cause deformities in the ventricular complex of the electro-

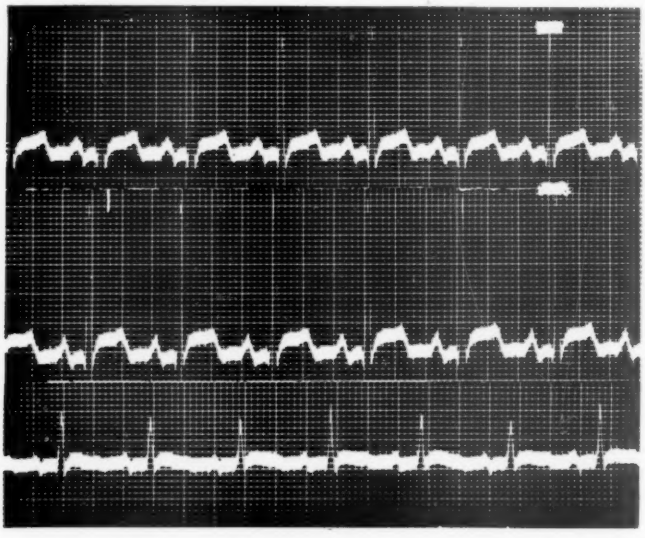

Fig. 5.—Curves from a case of rheumatic carditis, suspected of having a pericardial effusion. Note that the S-T segment is distinctly elevated above the isoelectric line and merges with an upright T-wave.

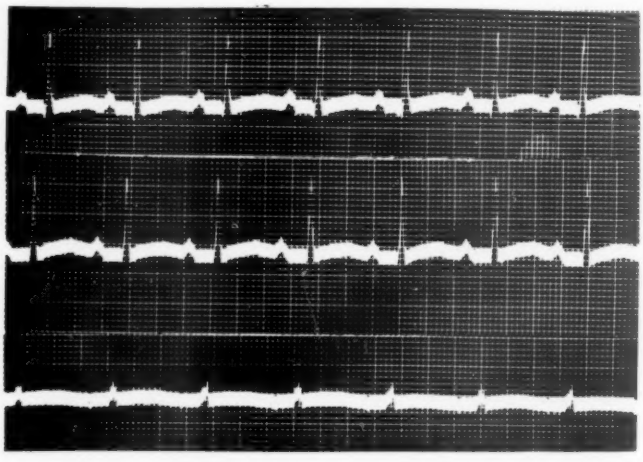

Fig. 6.—From the same patient nineteen days after Fig. 5. Note the return in the ventricular complexes toward normal.

cardiogram, but to accept this conclusion unqualifiedly seems unwarranted in the light of our observations. Furthermore, we are acquainted with no published case of rheumatic carditis showing definite

R-T deviation in which the question of pericardial effusion can be positively ruled out. Until such instances appear it seems to us that the question remains *sub judice*.

As stated above, we feel that our first case of hemopericardium is a clear example showing the effect of hydrostatic pressure in the pericardium on the ventricular complex of the electrocardiogram. The questions arise: First, what factors are concerned, and second, why is the R-T deformity in pericardial effusion so like that seen in recent myocardial infarction? These questions will be considered in detail in the paper dealing with our experimental observations on dogs. In brief, the functional effect of pericardial effusion is determined primarily by the hydrostatic pressure exerted on the heart—*Herztamponade*. This compresses the vascular channels and leads to anoxemia of the heart muscle. In addition the cardiac output is reduced, so that the coronary flow is impaired.

The hydrostatic pressure in the pericardium may vary widely in clinical cases because of such variable factors as (1) the element of time, i.e., the rate at which fluid accumulates in the pericardial sac, (2) the quantity of fluid—each further increment causing a greater elevation of hydrostatic pressure than the previous one, (3) the distensibility of the parietal pericardium—a given quantity of fluid in a rigid sac, e.g., tuberculosis of the pericardium exerting more pressure on the heart than the same quantity of fluid in a more elastic sac.

Referring now to the clinical curves, it is apparent that the later records from each case exhibit less deformity in the S-T segment than the earlier ones. In other words, as time elapsed the ventricular complexes became more normal in appearance. Assuming as we do, a direct relationship between hydrostatic pressure in the pericardium and anoxemia of the heart muscle, it follows that as the intra-pericardial pressure is lowered—by stretching of the parietal pericarduim or absorption and organization of the effusion—the anoxemic state of the heart muscle is relieved. On this basis the more nearly normal ventricular complexes found in the later records can be explained.

In conclusion we suggest that since the R-T deviation observed in coronary thrombosis occurs also in pericardial effusion, the term "coronary T-wave" is misleading and should, therefore, be discarded.

SUMMARY

Electrocardiograms from a case of hemopericardium and a case of purulent pericarditis with effusion are recorded which exhibit R-T deviations similar to those seen in recent myocardial infarction. These changes are ascribed to increased hydrostatic pressure in the pericardial sac which probably causes anoxemia of the heart muscle.

Observations on a case of rheumatic carditis showing abnormal ventricular complexes are included. In the interpretation of R-T deviation in rheumatic heart disease the presence of pericardial effusion must be considered.

The suggestion is made that since the R-T deviation observed in coronary thrombosis occurs also in pericardial effusion, the term "coronary T-wave" is a misleading one and should therefore be discarded.

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THE ELECTROCARDIOGRAM IN PERICARDIAL EFFUSION*

II. Experimental

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THE possibility that the electrocardiographic changes reported in our previous clinical paper are actually caused by pericardial effusion, was tested in experiments on normal dogs. For this purpose the experimental method described by Katz and Gauchat[1] was used. In brief, the dogs were anesthetized with morphine and barbitol, and artificial respiration instituted. A hole was made in the chest wall and a

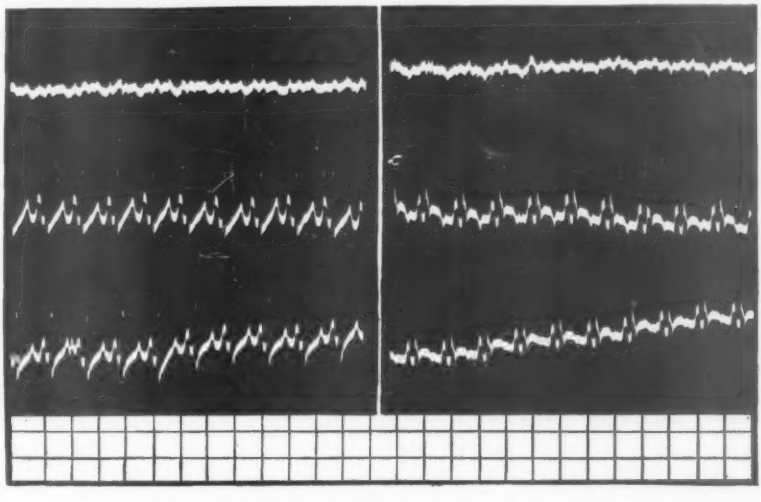

Fig. 1.—Electrocardiograms, three standard leads, showing the effect of acute experimental pericardial effusion. Segment on left, control; that on right, after 50 c.c. of isotonic saline were injected, and the pericardial pressure elevated to 220 mm. of saline. In this figure and succeeding ones, the scale at the bottom shows time in 0.2 second (ordinates) and voltage in 0.5 millivolts (abscissae).

specially constructed cannula was tied into the pericardium. Then the chest wall was repaired and a pleural cannula inserted, through which the pneumothorax was relieved. The cannula was now closed and normal respiration was resumed.

Electrocardiograms were obtained by the usual three leads and were standardized so that one centimeter was equivalent to one millivolt. The Victor electrocardiograph was used, its clockwork arranged to run 2.5 cm. per second. An electrocardiogram was taken after a certain amount of oil or isotonic saline had been forced into the pericardial sac by means of a 200 c.c. syringe, and the record compared with

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the control curves taken before the injection. The intra-pericardial pressure was registered with a U-tube manometer containing the same liquid used in the injection.

The principal changes observed in the electrocardiograms before and after experimental pericardial effusion are illustrated in Figs. 1, 2, 3, 4, and 5; in the legends of these figures are given the data concerning the amount of fluid injected and the resulting elevation in the pericardial pressure.

RESULTS

The following general effects are worthy of note. In some animals dyspnea was produced, in others apnea occurred following the pericardial effusion. In many instances the heart developed premature

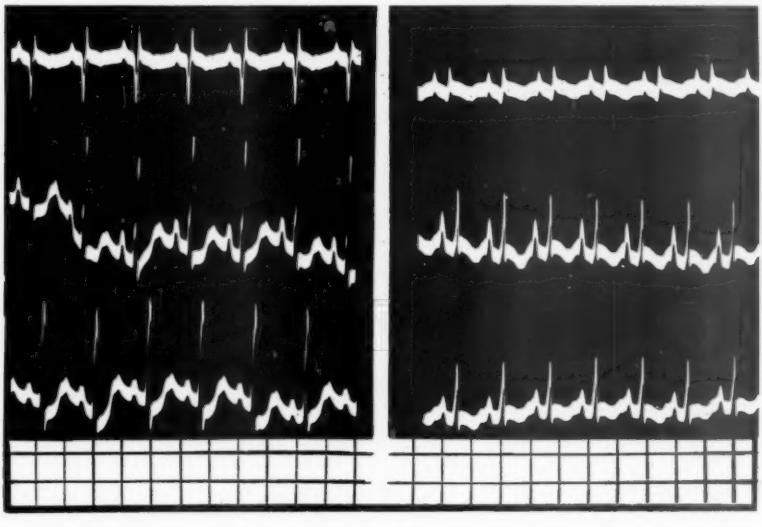

Fig. 2.—Three leads showing the effect of acute experimental pericardial effusion. Segment on the left is control; that on the right, after 70 c.c. of oil were injected into the pericardial sac and pressure raised 110 mm. of oil.

contractions, sinus bradycardia (see Figs. 3 and 4), or complete heart-block. These abnormalities disappeared as a rule when the fluid was removed. The pressure in the pericardial sac rose greatly, and as Katz and Gauchat[1] found, the respiratory undulations tended to disappear.

The electrocardiographic changes observed in the ventricular complex fall roughly into three categories:

1. The first type of R-T deviation resembles that found in our clinical cases and belongs to the so-called group of "coronary T-waves." It was found in four experiments on four animals out of a total of fifteen experiments on six dogs. The QRS complex becomes smaller in this type, the S-T segment does not shorten but is distinctly raised and is followed by a small inverted or upright T-wave. Three examples of this type are shown in all leads of Figs. 1 and 2, and in Lead II of Fig 3.

In Fig. 1, Lead I, the voltage is so small that the changes are minimal. The changes in Leads II and III resemble each other. In both, the voltage of the QRS complex decreases, and slurring develops near the top of the descent of the R-wave; the S-wave does not quite reach the

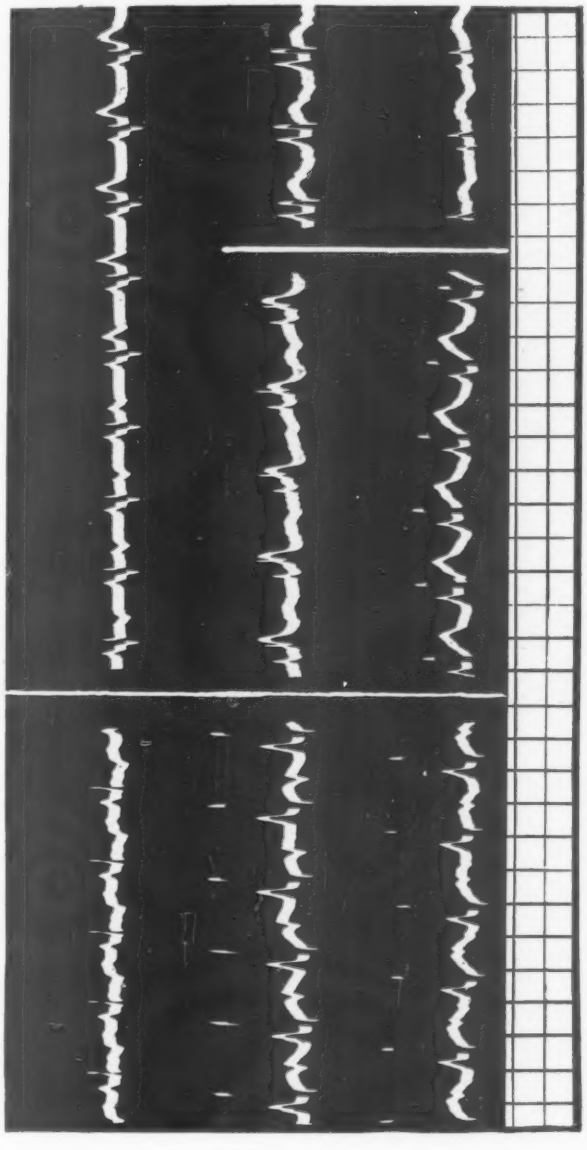

Fig. 3.—Three leads showing the effect of acute experimental pericardial effusion. Segment on the left, control; that in the middle, after 200 c.c. of oil injected and pericardial pressure elevated 165 mm. of oil. Note development of changes in Lead I, and also the different types of changes in the three leads. Segment on the right (Lead I omitted), after an additional 200 c.c. of oil were injected; the pericardial pressure now dropped to 180 mm. oil because a leak developed. The pressure was 140 mm. while Lead II was taken, and fell to 100 mm. as Lead III was recorded. Note the P-wave changes and the sinus bradycardia.

iso-electric level and is followed by a positive S-T segment, and this by a small negative T-wave. In Fig. 2 the changes in the S-T interval and T-wave of all three leads are similar to those of Leads II and III in Fig. 1. The QRS deflection becomes smaller in all leads; the negative

phase disappears in Lead I, becomes smaller in Lead III, and does not go below the iso-electric level in Lead II. In Fig. 3 the third segment of Lead II shows changes similar to those seen in Fig. 1. In the middle

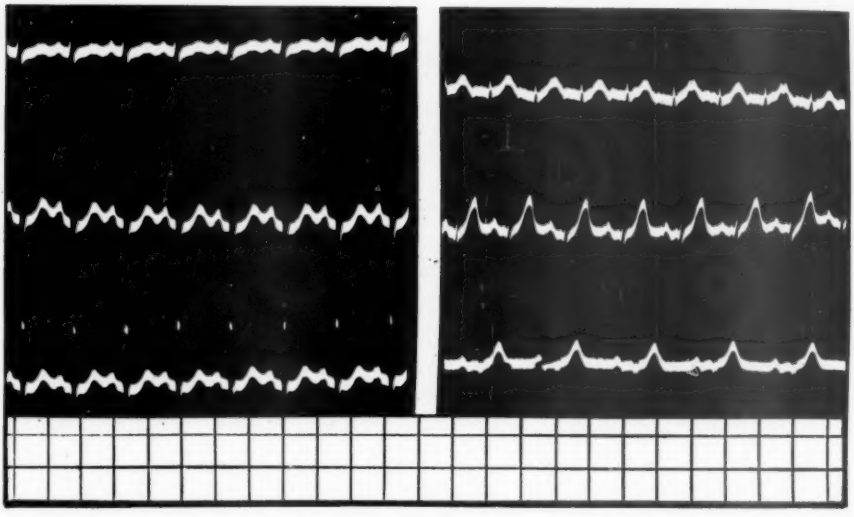

Fig. 4.—Three leads showing the effect of acute experimental pericardial effusion. Segment on left, control; that on right, after 180 c.c of isotonic saline injected and pressure in pericardial sac elevated to 320 mm. of saline. Note the P-wave changes and, in Lead III, the sinus bradycardia. White block in Lead II indicates height of R-wave in control record.

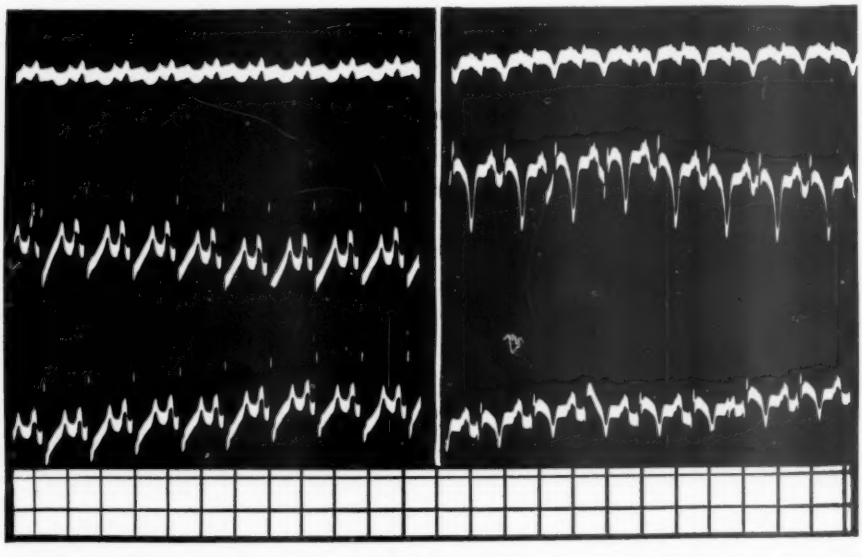

Fig. 5.—Three standard leads showing the effect of acute experimental pericardial effusion. Segment on left, control; that on right, after 80 c.c. of isotonic saline were injected and the intrapericardial pressure raised to 270 mm. of saline.

segment of Lead II (Fig. 3), the S-T interval is shortened and takes off high on the QRS group.

2. The second type of R-T deviation, shown in Leads I and III of Fig. 3, and in all leads of Fig. 4, was found in four experiments on two dogs. The QRS complex becomes smaller in this type also; the S-T interval shortens and its level remains unchanged or becomes slightly positive. The electrocardiogram is dominated by a rounded, broad, tall T-wave. The most typical change is seen in Fig. 4, especially in Lead II. Lead III of Fig. 3 shows two deviations of this type; in the last segment there is no shortening of the S-T interval and the T-wave is not very tall; in the middle segment the S-T interval is very short and the large T-wave is peaked. Lead I of Fig. 3 is very interesting because we were able to record the development of this type of change. It so happened that the record was taken inadvertently before all the fluid had been injected. The QRS takes on its final form in the first beat, but the T-wave is little changed; in the next three beats a small positive phase gradually develops in front of the negative one which gradually disappears. In the fourth beat a noticeable shortening of the S-T interval occurs which progressively becomes more marked in the succeeding beats, and the T-wave becomes more prominent.

3. The third type of R-T deviation is shown by eight experiments on two dogs. It is well illustrated in Fig. 5. The QRS complex becomes smaller in Leads II and III. The S-T interval does not shorten,* but becomes positive to varying degrees in the different leads. The dominant feature, however, is the deep inverted and peaked T-wave; note especially Lead II which is more typical of the usual change in this group.

It is significant that two types of deviation may occur in different leads of the same record, as in Fig. 3, or at different times in the same animal. For example, the curves in Fig. 1 and in Fig. 5 were obtained in different experiments on the same animal.

DISCUSSION

Such changes in the electrocardiogram as here recorded are not due to shifting in the axis of the heart, since the records of Cohn,[2] and Meek and Wilson[3] show no changes in the S-T interval or T-wave resembling those here described. Similarly, the relatively insignificant alterations found by Katz,[4] when the chest was opened and manometers inserted in the heart, rule out the possibility that the changes are due to short-circuiting. The decreased voltage of the QRS group was not due to insulation, as saline injections gave the same results as oil. It follows that these electrocardiographic changes must be due to the experimentally produced pericardial effusions, for they appear when the effusion is made and disappear when the effusion is removed. And

*In practically all the other instances, however, a distinct shortening of this phase is present.

the similarity of the experimental types to the clinical implies that the changes observed in our patients were in all likelihood due to the presence of fluid in the pericardial sac.

These experimental electrocardiographic changes, which resemble the clinical curves seen in recent occlusion of the left coronary artery can be explained on the basis that severe anoxemia of some regions of the left ventricle develops, causing a delay in the conduction of the impulse and a weaker response (mechanical and electrical) in the regions involved. In pericardial effusion the rapid accumulation of fluid in the pericardial sac causes an elevation of intra-pericardial pressure, which in our experiments reached in some cases 30 mm. of Hg. As shown by Katz and Gauchat[1] and by others, the pressure in the pericardium causes an elevation in the intra-ventricular, intra-auricular and venous pressures on both sides of the heart. In other words, with the tension of the heart musculature around zero, as happens during diastole, the wall of the heart is still under tension as a consequence of the hydrostatic pressure in the pericardium. An extra-vascular pressure is thus applied to the blood channels in the heart wall, causing a diminution in capillary flow such as appears normally during systole, as shown by Anrep.[5] Two other factors also tend to retard the blood flow through the heart. In the first place, an elevation of pressure occurs in the coronary sinus, as in the other veins emptying into the right auricle, and thus there results an increased resistance to outflow from the coronary capillaries. In the second place, the elevated pressure in the pericardium impedes the filling of the heart, thereby reducing the cardiac output together with the arterial blood pressure, i.e., the driving force of the coronary circuit is reduced. In short, pericardial effusion impairs the blood supply to the heart, causing a state of anoxemia, which apparently affects the left ventricle more than the right. This is suggested by the similarity of the curves in pericardial effusion to those seen in occlusion of the left coronary artery.

The variations in the experimental curves are probably due to differences in the location of the severe anoxemic areas of the left ventricle in the several experiments. Anoxemia does not affect the same regions to the same extent in different experiments, so that varying degrees of intra-ventricular block and decreased electrical response are combined. The combinations are such that roughly three groups of changes can be classified, as have been described.

SUMMARY

The effect of acute experimental pericardial effusion on the electrocardiogram was studied in normal dogs to ascertain whether changes would occur similar to those observed in our clinical cases reported in the preceding paper.

Three general types of abnormal ventricular complexes are classified. In all types the QRS complex becomes smaller in voltage. The groups differ in regard to the modification of the S-T segment and the T-waves as follows:

(a) The S-T segment in this group, which resembled the clinical cases, usually remained unchanged in duration but rose distinctly above the iso-electric level; the T-wave became small, usually inverted, but occasionally remained upright.

(b) The S-T segment in this group was shortened but usually remained at the iso-electric level, and the electrocardiogram was dominated by an upright, broad, tall and rounded T-wave.

(c) The S-T segment in this group was also shortened. As a rule, the level of this segment of the curve rose above the iso-electric level, but the striking feature was the development of a deeply inverted and peaked T-wave.

The hypothesis is suggested that the changes in the ventricular complexes of the electrocardiogram in experimental pericardial effusion are caused by anoxemia of the heart muscle which is more marked in certain regions of the left ventricle, leading to intra-ventricular block and diminished electrical response in the involved regions.

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ABNORMAL ELECTROCARDIOGRAMS IN PATIENTS WITH SYPHILITIC AORTITIS*†

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THE purpose of this communication is to present a detailed clinical and electrocardiographic study of 50 cases of syphilitic aortitis, some with and some without physical signs of aortic insufficiency. Certain differences in the electrocardiographic findings of these two groups have been discovered which are thought to be of diagnostic and prognostic importance. A similar series of 85 cases has been reviewed by Heimann,[1] who found 15 with downward T-wave in Lead II and 49 others with a "delay, or a delay and diminished amplitude" of T. Of his 85 records, 64 showed T-wave changes of some sort, though it is not quite clear what is meant by "delay" of T, and he did not specify how many showed this change alone. He called attention especially to the notching of QRS which was present in 25 of his cases (30 per cent), and which he believed was more commonly associated with syphilitic myocardial changes than with any other condition.

The patients selected for this study showed definite evidence of syphilis in a positive history of a chancre, a positive Wassermann reaction, x-ray evidence of aneurysm or diffuse dilatation of the aorta in a young individual, or definite syphilitic lesions in another part of the body or in the aorta at post-mortem examination. Some showed only two of these features, but many of them showed more than two. In the least definite case the evidence rested upon a dilated aorta, and although the Wassermann was negative, there was a cutaneous lesion which was positively diagnosed as syphilitic, by Doctor A. B. Cannon of the dermatological service.

The group showing aortitis without insufficiency, Group 1, consisted of 16 cases; 15 of these were males and the other a female. The group showing aortitis with aortic insufficiency, Group 2, consisted of 34 cases; 28 were males and 6 females. The age distribution in these groups is shown in Table I. It will be noted that the group with aortic insufficiency is on the whole composed of somewhat older individuals than the group without this lesion. The serological tests are shown in Table II. The blood Wassermann reaction was positive in 86 per cent of all the cases, but only 5 cases or 10 per cent were nega-

*From the medical services and cardiographic department of the New York City Hospital.

This report is part of the study of cardiovascular syphilis undertaken on behalf of the Committee for the Coordination of Investigation of the American Heart Association.

†Read at the Fifth Annual Scientific Session of the American Heart Association, Portland, Oregon, July 9, 1929.

tive both as to blood and as to spinal fluid serology. The symptoms of which the patients complained are shown for the two groups in Table III. In the group without aortic insufficiency the equal frequency of

TABLE I

AGE DISTRIBUTION

YEARS	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
32 - 39	6	4
40 - 49	6	13
50 - 59	2	15
60 - 68	2	2

TABLE II

SEROLOGY

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Blood Wassermann Positive	12	30
Blood Wassermann Negative	2	3
Blood Anticomplimentary	1	–
Spinal Fluid Colloidal Gold Luetic Curve	1	1

TABLE III

SYMPTOMATOLOGY

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Tumor of the Anterior Chest	3	–
Hoarseness	2	1
Pain in the Anterior Chest	7 (44%)	13 (38%)
Pain in Both Shoulders	1	–
Pain in the Left Shoulder	2	–
Pain in the Left Arm	1	–
Pain in Left Side of Chest	1	–
Pain in Right Side of Chest	2	–
Pain in Back	3	–
Pain in the Epigastrium	1	4
Dyspnea	7 (44%)	26 (77%)
Edema	3 (19%)	9 (27%)
Weakness	2	4
Palpitation	1	6
Cough	1	2
Hemiplegia or Aphasia	–	44
Pulsation in Neck	–	2
Dysphagia	–	1
Epistaxis	–	1
Choking Sensation	–	1

shortness of breath and pain in the anterior chest is interesting, but if pains in the shoulder, arms, sides of the chest and back are also considered, it will be seen that pain of one sort or another is by far the most frequent symptom in these patients. In the group with aortic insufficiency the predominant symptom is shortness of breath, although pain in the anterior chest occurs with considerable frequency.

Edema is also a frequent symptom in this group; it occurred nine times, or in 27 per cent of the cases. This shows how advanced are the cardiac symptoms when these patients come to the hospital for treatment. In Table IV only the chief complaint is considered. Here we see as in Table III that patients without aortic insufficiency complain chiefly of pain; those with it complain chiefly of shortness of breath, although pain sometimes accompanies this.

TABLE IV

CHIEF COMPLAINTS

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Pain in the Back	2	–
Pain in the Anterior Chest	6*	2
Pain in the Chest and Dyspnea	4	12
Dyspnea With or Without Edema	3	12
Weakness	1	1
Palpitation	1	
Hoarseness and Dysphagia	1	
Aphasia	1	
Hemiplegia	–	2
Cough and Palpitation	–	1
No Cardiac Symptoms	–	1†

*In two cases the pain radiated to the arms.
†Entered hospital for inoperable carcinoma of cervix.

TABLE V

PHYSICAL EXAMINATION

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Tracheal Tug	1	–
Palpable Tumor		
Anterior Chest	7	–
Posterior Chest	1	–
Stridor	2	–
Systolic Murmur at the Apex	4	19
Systolic Murmur at the Base	4	33
Aortic Diastolic Murmur	–	34
Aortic Second Increased	4	–
Blood Pressure Normal or Less	14	19
Blood Pressure Increased	2	15
Corrigan Pulse	–	14
Large Pulse Pressure	1	34
No Abnormality Except by X-ray	2	–

In Table V is an analysis of the chief findings on physical examination. In the group without aortic insufficiency, murmurs over the cardiac valve areas are infrequent, and in this group also a normal blood pressure is common. In the group with aortic insufficiency a systolic murmur at the base occurs in almost every case, as well as the diastolic murmur of aortic insufficiency. Table VI shows the results of the x-ray examination of these patients. Every patient with aortic insufficiency showed a diffuse dilatation of the aortic arch. Five of these patients also had aneurysm. Cardiac enlargement was diagnosed in all except

TABLE VI

X-RAY FINDINGS

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Aneurysm of Aorta	11*	5
Diffuse Dilatation of the Arch	4	34
Cardiac Enlargement	8	33
"Duck-back" Appearance Typical of Aortic Insufficiency	–	22
Heart Not Enlarged	8	1†
Enlarged to the Right	2	18

*One case was not examined by x-ray, but showed aneurysm of the innominate artery at autopsy.

†This heart was reported "not enlarged but suggesting concentric hypertrophy."

TABLE VII

ELECTROCARDIOGRAPHIC FINDINGS

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Premature Beats	4	3
Auricular Fibrillation	–	3
Prolonged A-V Conduction Time	1	1
Right Axis Deviation of QRS	1	–
Left Axis Deviation of QRS	9 (51%)	31 (91%)
Neither Right nor Left Axis Deviation	6 (38%)	3 (8 %)
QRS Group		
Abnormal Duration	1	10
Notched or Slurred	3	10
Low Voltage	1	–
Unusual Peculiarity	2	2
High Voltage	2	11
Total Significant Abnormalities	6 (38%)	14 (41%)
T-Wave		
Downward in Lead I	4	9
Downward in Lead II	–	2
Downward in Leads I and II	–	15
Diphasic in Lead I	1	1
Diphasic in Leads I and II	–	2
Low Voltage	1	–
"Coronary" Type	1 (7 %)	8 (21%)
Total Abnormal T-Wave	6 (38%)	29 (85%)
No Significant Abnormality of QRS or of T	6 (38%)	5 (15%)

one of this group, but this one patient was said to show a concentric hypertrophy by the exaggeration of the left ventricular curve. In the group without aortic insufficiency cardiac enlargement was only diagnosed in half of the cases.

Table VII presents the electrocardiographic findings in the two groups. It will be noted that auricular fibrillation occurred only in the group with aortic insufficiency, and that many cases in the other group showed neither right nor left axis deviation of QRS. Left axis deviation of QRS was much more frequent in the group with aortic insufficiency, and 85 per cent of these patients showed an abnormality of QRS or of T which was considered significant of myocardial damage.

Only 62 per cent of those without aortic insufficiency had significant abnormalities in their records.

Abnormalities of the QRS group occurred with about equal frequency in those with and those without aortic insufficiency. Heimann[1] unfortunately did not describe the exact features of the notching of QRS which he found in 30 per cent of his series, and to which he attached so much diagnostic importance. Our series, however, showed only 7 records which resembled the notch of his illustration. Notching of the usual sort occurred in 13 of the records of our series, which is 26 per cent, or about the same frequency of occurrence as he described for the special form of notching. Notching occurred in 20 per cent of our cases without aortic insufficiency, and in 30 per cent of those with the lesion. Abnormalities of the T-wave occurred, however, in 85 per cent of those with aortic insufficiency, and in only 38 per cent of those without this lesion. The T-wave was of the "coronary" type[2] in 21 per cent of the group with insufficiency, and in only one case, 7 per cent, of the other group.

Seven patients without aortic insufficiency and 12 with it failed to recover. The mode of death is shown in Table VII-A. Although cardiac decompensation was the predominant cause in both groups, yet it was of much greater frequency in the patients with valvular disease. The deaths in the group with aortic insufficiency all occurred in patients who showed an abnormal T-wave in the electrocardiogram. All of the patients in this group who had normal T-waves recovered sufficiently to be discharged from the hospital, and it is possible that their recovery depended, in part at least, upon the absence of serious coronary or myocardial involvement as suggested by the normal T-wave.

Autopsies were obtained in 4 out of 12 of those patients with aortic insufficiency who died in the hospital, and on 6 out of the 7 without the lesion. The autopsy findings are shown in Table VIII. It will be seen that 2 patients who had aortitis but did not have physical signs of aortic insufficiency, showed a thickening of the valve cusps.

TABLE VII-A

MODE OF DEATH

	GROUP 1	GROUP 2 (AORTIC INSUFFICIENCY)
Decompensation	3	8
Bronchopneumonia	1	2
Anginal Attack	–	1
Carcinoma of Cervix	–	1
Tracheal Pressure	1	–
Rupture of Aneurysm	2	–

It appears from this that the valves must be involved to a certain definite degree before they become incompetent. Just as in the x-ray study, so also the autopsy revealed that half of the hearts without the

valvular lesion were enlarged, and half were of normal size, while in the group with the valve lesion, all were enlarged. Syphilitic narrowing of the mouths of the coronary arteries was observed in each patient with aortic insufficiency but was not found in any of those without this valve lesion.

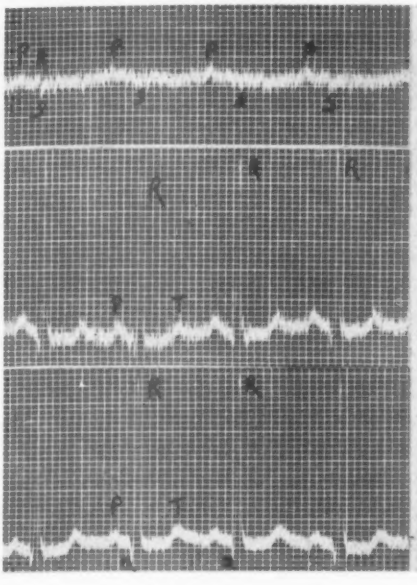

Fig. 1.-*A*.

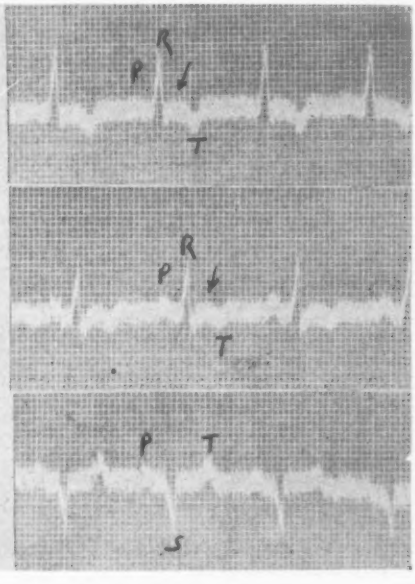

Fig. 1-*B*.

Fig. 1.—Electrocardiograms of Case 2, *A*, and Case 7, *B*, of the autopsy series Tables VIII and IX. Note in *A* the normal appearance of the electrocardiogram with a borderline right axis deviation of QRS, and in *B* the notching of the QRS group and the inversion of T in Leads I and II, with the coronary feature—the upward convexity indicated by the arrow—in both of these leads.

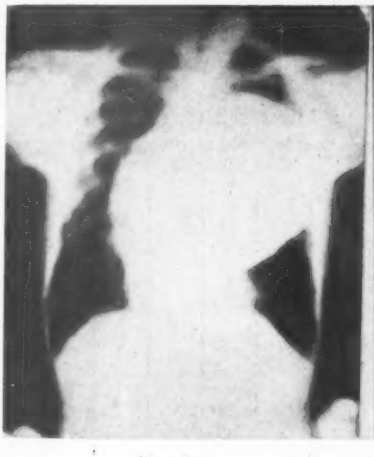

Fig. 2-*A*.

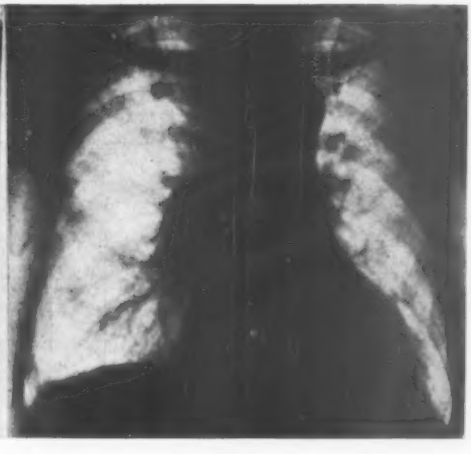

Fig. 2.-*B*.

Fig. 2.—Teleroentgenograms of Case 2, *A*, and Case 7, *B*, of the autopsy series, Tables VIII and IX. Note in *A* the small heart and the large aneurysm which involved the ascending portion of the arch. In *B*, note the dilated and somewhat tortuous aortic arch.

It will be interesting to consider the electrocardiographic abnormalities which were found in records from the autopsied cases. These are shown in Table IX. It will be noted that Cases 4, 5, and 6 of the group without aortic insufficiency showed an abnormality of the T-wave. No pathological changes were found to explain this abnormality in Case 4, but Case 5 showed arteriosclerosis of the coronary arteries and marked vascular renal disease. Case 6 also showed marked vascular renal disease, and it was thought that in these two cases the T-wave abnormality might be due to arteriosclerotic changes in the smaller coronary branches. All of the cases with aortic insufficiency showed T-wave abnormality. Case 9 also showed marked vascular renal dis-

TABLE VIII

AUTOPSY FINDINGS

CASE	1	2	3	4	5	6	7	8	9	10
Syphilitic Aortitis Without Aneurysm						x		x	x	x
Syphilitic Aortitis With Aneurysm	x	x	x	x	x		x			
Aortic Valves, Normal	x	x	x	x						
Aortic Valves, Thickened					x	x	x	x	x	x
Aortic Insufficiency							x	x	x	x
Syphilitic Involvement of Coronary Mouths							x	x	x	x
Marked Vascular Renal Disease					x	x			x	
Arteriosclerosis of Coronaries Without Occlusion	x				x					
Heart Enlarged			x		x	x	x	x	x	x
Heart not Enlarged	x	x		x						
Brown Atrophy	x	x								
CAUSE OF DEATH										
Rupture of Aneurysm	x		x							
Bronchopneumonia		x								
Tracheal Pressure				x						
Cardiac Failure					x	x	x	x	x	x

TABLE IX

ELECTROCARDIOGRAPHIC FINDINGS IN AUTOPSIED CASES

CASE	1	2	3	4	5	6	7	8	9	10
Premature Beats				x	x					
Prolonged A-V Conduction			x							
Right Axis Deviation of QRS		x								
Left Axis Deviation of QRS	x			x	x		x	x	x	x
Neither Right nor Left			x			x				
QRS Group										
Abnormal Duration							x	x		x
Notched or Slurred					x		x	x	x	x
Low Voltage			x							
High Voltage		x							x	x
T-Wave										
Downward in Lead I				x	x	x				x
Downward in Leads I and II							x	x	x	
"Coronary" Type					x		x			
No Significant Abnormality of QRS or of T	x	x								

ease, but the other 3 did not. It is believed that the T-wave changes of these 3 cases were due to the syphilitic narrowing of the mouths of the coronary arteries, which was demonstrated at autopsy.

Syphilis produces characteristic changes in the aorta. These may be described briefly as an infiltration about the vasa vasorum of the adventitia and media by wandering cells of the character of lymphoid and plasma cells with sometimes multinucleated giant cells. The elastic fibers of the media become necrotic, and diffuse scarring of this coat takes place. The intima becomes thickened and longitudinally wrinkled, and the entire vessel wall is thinned and weakened to a considerable extent, the end-result being either a diffuse dilatation or aneurysmal sac formation. This process usually begins in the ascending aorta, a few centimeters above the aortic ring, and spreads both upward and downward. As the process advances the mouths of the smaller branches of the aorta are inevitably encroached upon. It is evident from Cases 5 and 6 of our autopsied series that the process may descend far enough to involve the aortic valve, and yet the coronary orifices may not be grossly narrowed. Nine such cases were found by Clawson and Bell[3] in a group of 126 autopsies on patients with syphilitic aortitis. It is also conceivable that the coronary orifices might become involved when the aortic valves were still at least approximately normal. Such a case has been reported by Dr. H. S. Martland, associated with a congenitally high position of both coronary arteries, and 21 cases were found by Clawson and Bell in their series. Coronary narrowing by luetic aortitis must lead to a defective nutrition of the heart muscle, and this we believe is the cause of the abnormalities found in the electrocardiograms of our patients with aortic insufficiency.

The careful histological studies made by Clawson and Bell[3] of a group of patients clinically similar to ours revealed a narrowing of one or both coronary orifices in 22 of 28 cases with aortic insufficiency (79 per cent), but a study of the myocardium of these 28 cases revealed only microscopic fibrosis or proliferative reaction in 11 (39 per cent). Since we have found changes in the T-wave in 85 per cent of such cases, and histological changes are found in only 39 per cent, it is evident that the T-wave abnormality must often depend upon changes in the muscle physiology due to coronary narrowing but without demonstrable histological basis. These authors found narrowing of the coronary orifices in 3 of 23 patients with aneurysm of the aorta, and in 4 others of the 23 there was either microscopic fibrosis or a proliferative reaction. This is a much smaller incidence of both coronary and myocardial damage than in the group with aortic insufficiency, and is so small as to make us wonder why we found 62 per cent of abnormal electrocardiograms and 38 per cent incidence of T-wave abnormality in our group without aortic insufficiency. Here again the T-wave abnormality may depend upon myocardial changes which do not have a demonstrable pathological basis.

SUMMARY

Of 50 patients of syphilitic aortitis which were studied, two-thirds had aortic insufficiency and one-third did not; about one-third had aneurysm; 5 had both aortic insufficiency and aneurysm.

In general the patients with aortic insufficiency were older than those without; shortness of breath was their chief complaint, though pain in the anterior chest was frequent, and almost one-third complained of edema. All but one showed a systolic murmur at the aortic area.

The electrocardiogram showed an abnormal T-wave in 85 per cent of these patients, and in 20 per cent it was of the "coronary" type. It was abnormal in only 38 per cent of those without the valve lesion, and only 1 case (7 per cent) showed a wave of the "coronary" type.

Ten autopsies were obtained on these 50 cases, and from a study of the autopsy material and the electrocardiographic records it appeared that the abnormality of the T-wave is probably due to encroachment upon the lumen of the coronary orifices by the syphilitic disease in the sinuses of Valsalva. The greater frequency of the T-wave changes in the group with aortic insufficiency is due to the fact that in these patients the aortitis involves the region of the valves near which the coronary arteries originate.

Changes in the T-wave of patients with syphilitic aortitis should be viewed as an indication of serious coronary involvement, but not necessarily as an indication of myocardial pathology.

This observation has an extremely important bearing upon our general understanding of the causes of abnormality of the T-wave.

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(For discussion, see page 115.)

SYPHILITIC CORONARY OCCLUSION IN AORTIC INSUFFICIENCY*†

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IMPAIRED circulation resulting from disease of the coronary arteries causing symptoms, heart failure and death is receiving an increasing amount of attention since Obrastzow and Straschezko in 1910,[1] and Herrick in 1912,[2] called attention to the syndrome accompanying the sudden occlusion of these arteries. Since this condition is being recognized more generally by the profession, the frequency with which it occurs indicates that it is probably a far more common lesion than has been suspected in the past. Those factors, therefore, having a causal relation to coronary disease, whether predisposing to the precipitation of clots, proliferation of the intima, atheroma, or any other change becomes of prime importance. Syphilis has long been recognized as causative in certain cases. It is this condition that I will discuss, presenting two case reports and a syndrome which I believe will permit of recognition in certain cases.

The first observations on coronary disease seem to have been made by Drelincourt (1700),[3] and the association of coronary and myocardial disease was first noted by Bellini (1703).[3] Later Edward Jenner[4] and also Parry[4] believed disease of these arteries to be the underlying lesion in angina pectoris, though Allbutt insisted that what Jenner really said was that, in patients dying of angina, coronary sclerosis would be found, which he felt was quite a different matter.

Involvement of the aorta by syphilis, resulting in aneurysm [a relation suspected by Paré[5] and later insisted upon by Lancisi (1728) and Morgagni (1761)[5] and established by Welch, (1876), Dohle (1888)[5] and confirmed by others] and also the deformity and destruction of the aortic valve resulting in insufficiency (so well described by Dominick Corrigan in 1832)[6] are common conditions in the southern negro. The combination of aortic insufficiency with coronary closure is commented upon by most authors as of frequent occurrence. Benson states that syphilitic occlusion accounts for most closures of the coronaries and that the effect on the heart is similar to other gradual closures of these vessels where sufficient time has elapsed for anastomosis with the other coronary to develop. According to Stokes,[7] in

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advanced grades of involvement of the aortic valve the encroachment on the coronaries may be expected to be severe. The symptoms may not be a guide, for even in mild grades of aortitis coronary change may be so severe as to menace life.

Osler in his Lumelian Lecture[8] stated, "A man may get on very comfortably with practically a fourth of the whole coronary system," and Allbutt[9] goes even farther and says that, "a man may get on with the whole system occluded, so far, at any rate, as the orifices and main trunks are concerned," and concluded with Kanthack that other things being equal, the factor of safety is the rate of occlusion.

The following case reports illustrate certain features of interest:

CASE REPORTS

CASE 1.—E. E., well-nourished colored female, 26 years old, admitted January 22, died January 24, 1927. Stated that three weeks prior to admission she developed a cough, slight sore throat and soreness through her chest. One week later she developed shortness of breath and swelling of feet and legs. She had leucorrhea but denied venereal lesion. History otherwise not relevant.

Physical Examination.—Temperature 97°; pulse ranged between 88-130; respiration 28-44, labored and shallow; blood pressure, systolic 120 mm., diastolic 50 mm.; mucous membranes of mouth and throat congested; veins of neck prominent; râles in lower lobes of both lungs posteriorly; cardiac dullness to sixth interspace, 12.5 cm. to left of midsternal line. Apex not visible, soft systolic murmur at apex poorly transmitted. No signs of fluid in abdomen, which was distended and tympanitic. Some swelling of feet and ankles. Hemoglobin 50 per cent. Red blood cells 2,100,000, leucocytes 9,000, 52 per cent polynuclears; catheterized urine—acid, specific gravity 1.010. Acetone 1 plus, otherwise negative. Wassermann, Kolmer antigen, plus 4.

Autopsy.—This subject was a young negro woman, about 26 years old, about 5 feet tall, weighing about 100 lbs., in well-nourished condition.

There was a moderate anasarca, most noticeable in the legs and neck and general evidence of chronic heart failure in general marked passive congestion, edema, tissue degeneration and fibrosis, this being particularly in evidence in the lungs, liver and spleen. The kidneys showed merely cloudy swelling. The uterus was infantile, and there were bilateral dermoid cysts of the ovaries, each the size of a large orange. There was no arteriosclerosis.

The aorta was pliable and elastic everywhere except in two areas. One of these was just distal to the aortic orifice and included a part of one of the aortic valve cusps. The other was up in the arch of the vessel. These areas were quite similar in appearance, the proximal one being roughly pear-shaped and occupying about one square inch of intimal surface. It began just behind the left posterior cusp, involving this cusp, and through its center coursed the completely obliterated mouth of the left coronary artery. The higher area was roughly rounded and about twice the size of the first. These areas were raised above the intimal level almost one fourth of an inch, were firm and of cartilagenous consistency. They were of a hyaline pale bluish appearance depressed and yellowish in the center. Over their surfaces were rough lines generally paralleling the course of the vessel. There were a few yellow atheromatous patches at other points in the intima. Microscopically, these areas are of typical syphilitic aortitis, with prominent vasa vasorum in the outer coats surrounded by lymphocytes, patches of necrosis in the media and marked hyaline fibrous thickening of the intima.

The heart was extremely soft and flabby, a collapsing "dish-rag" heart. The cavities were dilated but there was little or no hypertrophy. On section the muscle wall could be seen to have fibrous strands through it. There was slight thickening of the margins of the mitral leaflets. One cusp of the aortic valve was thickened, stiff, retracted and bound down into the syphilitic lesion about its base, as described above, giving definite valvular insufficiency. Microscopically some of the muscle of the heart wall showed definite, but not outstanding hypertrophy. There was general fibrosis, congestion and parenchymatous degeneration. Near the endocardium was extreme parenchymatous and fatty degeneration and fibrosis. The coronary vessels were open except as described above at the mouth of one main artery.

The disability in this case plainly was the result of the syphilitic lesion at the aortic ring, giving slowly progressive aortic insufficiency and at the same time, gradually closing the mouth of the involved coronary artery. This nutritional impairment of the heart prevented the ordinary response of cardiac hypertrophy and led to a failure of the heart, unusually rapid in progress to completeness.

CASE 2.—M. G., Colored female, 26 years old, entered October 14, died, October 19, 1927. For three months, she had noticed palpitation and shortness of breath aggravated by exertion. Forced to stop all work two weeks prior to admission. Feet, legs and abdomen began swelling ten days before admission. Three children living and well, no miscarriages. No history of initial sore. History otherwise not relevant.

Physical Examination.—Temperature ranged from 96°-99°, reaching 100.5° the day before death. Pulse averaged 120. Respiration averaged 26 but was labored and shallow. Blood pressure 114 mm. systolic, 46 mm. diastolic. Heart not apparently enlarged; apex in fifth space 8 cm. to left of midsternal line. Thrill over precordium corresponding to to-and-fro murmur; moderate general edema. Hemoglobin 75 per cent, red blood cells 3,800,000; white blood cells 34,000; polynuclears 80; Wassermann plus 4; catheterized urine—acid, specific gravity 1.010, albumin plus 1; casts, hyaline plus 1; finely granular plus 2; otherwise negative. Blood culture negative.

Autopsy.—This subject was a negro woman about 26 years of age, of good development and state of nourishment and with marked general edema, anasarca, ascites and some excess fluid in pleural and pericardial cavities. There was general evidence of chronic heart failure in passive congestion, edema, tissue degeneration and fibrosis, this being prominent in lungs, liver, spleen and kidneys. There was a well-marked sclerosis with calcification of the walls of the small arteries in the uterus, and this organ was fibrous. The ovaries contained several small cysts. The kidneys showed some sclerosis of small arteries with some glomerular fibrous obliteration, patchy cortical fibrosis with lymphocytosis.

In the first part of the aorta, involving two cusps of the aortic valve and the mouth of the right coronary artery was an elevated, bluish, hyaline plaque, with rough and striated center. The valve cusps were thickened, uniformly shortened, retracted, and were agglutinated to each other at their adjoining ends. The mouth of the involved coronary was completely closed and the lumen obliterated by fibrosis to the first branch. The remainder of the coronary system was open. Section of this area in the aortic wall showed characteristic syphilitic aortitis, with fibrous thickening and surrounding lymphocytosis of the vasa vasorum, degeneration, necrosis and pronounced fibrosis of media and intima.

The heart was little if at all hypertrophied, some of its fibers being large on section. The musculature was extremely flabby and pale, and the cavities were

dilated. The muscle fibers generally were granular and near the endocardium very markedly vacuolated. Here particularly, were marked congestion and fibrosis. The capillaries generally were engorged with blood and along their courses were many polynuclear leucocytes. There were some diffuse fibrosis and noticeable mononucleosis. Beneath the epicardium, especially at the base posteriorly, were numerous petechial hemorrhages.

This heart could be reasonably called that of chronic myocarditis, or carditis, since all its parts were involved in a process which was more than the ordinary degeneration and fibrosis. While the state may be one of syphilitic carditis, it appears that the main condition and disability was the result of the syphilitic lesion at the aortic ring with resulting aortic valve insufficiency and a coincident occlusion of the mouth of one coronary artery, again so handicapping the organ as to bring about a progressive failure to completion in a period of time and with a rapidity of development of signs of cardiac failure different from simple aortic insufficiency, even of such origin.

These two cases were selected as the basis for this report because of their striking similarity in so many features. In each case the age was 26 years. While it is generally accepted that the time elapsing from the chancre to the development of the aortic leak averages from 18 to 20 years, it is also well known that this is not always the case. Brooks[10] reports 5 cases in patients under 30 years of age and states that they showed a most striking and extensive coronary involvement.

The patients here reported were both women. In a series of 146 consecutive cases recently admitted to the cardiovascular service of Roper Hospital, there were 83 males and 19 females. Both of the cases reported here were colored. The course was rapid in each case, 3 weeks and 3 months respectively. It is possible that the patients may have been sick longer than they stated, and that they dated the onset from the time they noted disabling symptoms. However, as Allbutt[11] states, "For I would repeat that the course of cardio-aortic syphilis combined, as it too often is, with coronary disease may be stealthily swift. Indeed, it is a kind of microbic endocarditis." Brooks[12] records a case of perforation of one of the coronary sinuses before the secondary rash appeared.

The systolic blood pressure was not elevated, but the diastolic was low, resulting in an increased pulse pressure, 114-46 in one, and 120-50 in the other. The respiration in each of these cases was notable for being labored rather than for rapidity, averaging around 27 in one case, and 35 in the other.

The Wassermann reaction was positive (plus 4) in each and was 89 per cent positive for the group of 38 colored women in our series, while for the 6 white women of the group, there were only 33 per cent positive reactions. The percentage of positive reactions for the group of 146 cases was 82 per cent, 48 per cent for the white and 89 per cent for the colored.

The post-mortem examination disclosed syphilitic aortitis with aortic insufficiency in each case. Allbutt[13] states that aortic valve involvement would appear to be present in about 30 per cent of all cases of syphilitic aortitis, mild and severe. Arneth in 202 cases of tertiary syphilis states he found 3.2 per cent of aortitis and 2 per cent of aortic regurgitation. In our series, the diagnosis of incompetency of the aortic valve was made 51 times in about 35 per cent of all cases *or* of cases with aortitis. Some of these cases were readmissions, but if we take the actual number of new cases, 107, aortic insufficiency was present in 23 cases (21 per cent).

Both these cases had a main branch of the coronary occluded at the ostium; in one, the right branch, and in the other, the left. All authors agree that this is common in aortitis, though I could not find any figures quoted. G. A. Allen,[13] in analyzing 1,000 consecutive post-mortems, found macroscopic lesions of the coronaries in 371 cases. In 97, there was definite narrowing or blocking of the lumen, only 7 of which were due to syphilis. According to Allbutt[11] the right is usually first involved, and he quotes Broadbent as insisting that this is the reason that cases of syphilitic aortic insufficiency do badly. Clinically, there was no appreciable difference in the two cases suggesting any variation depending on which artery is occluded. The patient with blocking of the left artery was sick only three weeks and showed much more congestion of the mucous membranes of mouth and throat and dilated neck veins; the one with the occlusion of the right branch was sick three months and showed more anasarca and ascites.

Each of these two cases failed to exhibit the usual response to incompetency of the aortic valve with hypertrophy. Whether this was due to the lack of a normal myocardium as postulated by Krehl[14] or to the lack of adequate nutrition due to blockage of the coronary is questionable. Personally, I think the latter is more likely and that the impaired nutrition itself contributed to a more rapid degeneration of the muscle and the absence of hypertrophy. It is regrettable that owing to the stress of routine, these sections were not stained for the spirochete. There is much in the picture to suggest that they would have been found.

SUMMARY

Two cases of syphilitic aortitis with insufficiency and occlusion of a main branch of a coronary artery are reported. The striking similarity of the cases is commented upon, and statistics from analysis of a small series of cases are utilized.

It would seem therefore that in young adults with syphilis and aortic insufficiency who do not exhibit the usual compensatory hypertrophy and whose progress is rapidly toward a fatal outcome, one may reasonably presume the involvement of one or more of the coronary openings in the syphilitic process.

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(For discussion, see page 115.)

THE INCIDENCE OF HEART DISEASE IN THE PACIFIC NORTHWEST*

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With increasing interest in heart disease it is apparent that certain cardiovascular lesions may be more prevalent in one part of the country than in another. It has been shown that acute rheumatic fever is more prevalent in rigorous climates than in equable ones.[1, 2, 3] Syphilis in a charity hospital in the South is more common (largely because of negro admissions) than in other localities.[4] In regions where goiter is endemic the cardiovascular symptoms due to this condition can be studied to advantage. One would expect the more common forms of heart disease, such as those associated with arterial hypertension, to be equally distributed regardless of locality. A comprehensive report of the work recently carried out in New York State[5] serves as an example of the value of local studies.

In attempting a study of the incidence of heart disease in the Pacific Northwest, we are aware of the size of the problem and the inadequacy of material for careful and comprehensive statistical value but have obtained, we think, a preliminary cross-section of the material at hand.

For comparison with other parts of the country as to mortality from all causes and mortality due to diseases of the circulatory system, the following tables are presented:†

Table I

Death Rate Per 1000 Due to All Causes

OREGON	CALIFORNIA	WASHINGTON	MAINE	NEW YORK
Average for 10 yrs. to 1927	Average for 9 yrs. to 1926	Average for 9 yrs. to 1926	Average for 9 yrs. to 1926	Average for 9 yrs. to 1927
11.4	14.2	10.52	13.8	13.7

Table II

Death Rate Per 1000 Due to Disease of the Circulatory System

OREGON	CALIFORNIA	WASHINGTON	MAINE	NEW YORK
Average for 10 yrs. to 1927	Average for 9 yrs. to 1926	Average for 7 yrs. to 1926	Average for 7 yrs. to 1926	Average for 7 yrs. to 1926
1.9	2.62	1.62	2.77	2.74

*Read at the Fifth Annual Scientific Session of the American Heart Association, Portland, Oregon, July 9, 1929.

†From the Oregon State Board of Health.

MATERIAL STUDIED

To obtain a cross-section of data for study composed of patients presenting themselves in private practice and in hospital admissions the following sources were used, comprising a total of 28,661 cases, of whom 13,258 were medical patients. While these figures are all obtained from one locality in the Pacific Northwest, many of the patients came from adjacent states. To make the figures more comprehensive, figures from the larger cities of the state of Washington are desirable. The relation of cardiovascular disease to total admissions and to medical admissions is shown in Table III.

TABLE III

TOTAL ADMISSIONS	MEDICAL	CARDIOVASCULAR DISEASE		RHEUMATIC HEART DISEASE				GOITER	
				ACUTE		CHRONIC			
			Per cent of medical patients		Per cent of medical patients		Per cent of medical patients		Per cent of medical patients
Private patients 5,489	5,489	1,673	30	9	0.10	125		113	8.6
Multnomah patients 3,851	1,945	1,210	62	0		325		44	
U. S. Veteran Hospital 1,003	445	151		0		0		20	
Good Samaritan Hospital 8,452	3,650	208	5	3		57		161	
St. Vincent's Hospital 8,590	1,318	230	18	4	0.11	45		479	
Doernbecher Memorial Hospital for Children 1,276	417	16		6	0.12	0		4	
Totals 28,661	13,258	3,488	26	18	0.11	552	4.9	821	6.1

The Multnomah County Hospital is the teaching hospital of the University of Oregon Medical School and is affiliated with the Out-patient Department, the Portland Free Dispensary. The patients are mostly past middle age: therefore the incidence of cardiovascular disease is high (60 per cent). Patients with chronic valvular disease are also probably more numerous than in other hospitals. The Good Samaritan and St. Vincent's hospitals are private institutions, the majority of patients being surgical (59 per cent, Good Samaritan; 86 per cent, St. Vincent's).

Of the 13, 258 medical patients, 3,488 or 26 per cent showed cardiovascular disease. Acute rheumatic fever shows a low incidence, which suggests error. In the private patient group but few children are seen, which would lower the figure, but the Doernbecher Hospital for Children shows only a slightly higher figure. Pediatricians in the Pacific Northwest are of the opinion that the disease is quite rare, though no

published studies are available. Here the incidence of acute rheumatic fever was 0.12 per cent. Climatic factors, as brought out by the authors referred to,[1, 2, 3] may be assumed to explain this, for the climate of this territory is mild.

Faulkner and White[1] found the incidence of rheumatic fever and chorea to vary from 0.2 to 5.8 per cent of medical cases. Chronic valvular disease, as seen in their study, is no criterion as to incidence, for many of these patients date their trouble to rheumatic fever in childhood. They are not natives of this part of the country. Goiter in this region is endemic. The figures take into account only goiter patients in whom circulatory symptoms were prominent.

CLASSIFICATION OF VARIOUS TYPES OF HEART DISEASE

While hospital records were found satisfactory for the above, they were disappointing for use as to structural or etiologic classification because of methods of indexing diseases. One finds such ambiguous terms as "heart failure" and "broken compensation" and "dilata-

TABLE IV

CLASSIFICATION OF VARIOUS TYPES OF HEART DISEASE OF PRIVATE PATIENTS, ETIOLOGICAL AND STRUCTURAL, TO CONFORM WITH OTHER REPORTS

CLASSIFICATION	TOTALS			PER CENT OF TOTAL CARDIOVASCULAR DISEASE
"Heart pain" group	237			
From syphilitic group		1		
Total			238	14.2
Goiter, producing cardiac symptoms	133			
With fibrillation		10		
With flutter		1		
Total			144	8.6
Rheumatic heart disease (including 5 cases of rheumatic fever, and 1 case of subacute bacterial endocarditis)	134			
With fibrillation		34		
With heart-block		1		
Total			169	10.1
Hypertensive cardiovascular disease (including 33 cases showing hypertensive menopause symptoms)	604			
Heart pain and hypertensive cardiovascular disease		22		
Auricular fibrillation and hypertensive cardiovascular disease		1		
Total			942	56.3
Syphilis of the cardiovascular system	70			4.1
Irritable heart	285			17.0
Pericarditis (including 1 case of Pick's disease)	3			0.1
Auricular fibrillation	150			8.9
Auricular flutter	2			0.1
Dextrocardia				0.06
Functional murmurs (congenital?)				2.6
Auriculoventricular block	9			0.5
Total	1,673			

tion'' with no cross index as to type of heart disease present. Nor is it possible to get accurate figures as to hypertensive cardiovascular disease for these are classified as ''arteriosclerosis,'' ''hypertension,'' ''apoplexy'' or ''hemiplegia.'' We venture to say that this condition is not peculiar to these hospitals which are all class-A institutions.

We should like to know the incidence of heart disease in surgical patients, or what percentage of elderly patients with pneumonia shows evidence of cardiovascular disease.

Records of 1,673 private patients with cardiovascular sypmptoms were studied as to etiological, structural and functional diagnoses. The results are shown in Table IV.

DISCUSSION

In the ''heart pain'' group we are aware of the difficulty of separating these into coronary occlusion, coronary sclerosis, aortitis, etc., because of confusion in differentiating them as noted by us in a former paper.[5] In all these patients, however, pain was brought on by effort and relieved by vasodilators. Electrocardiographic records were often typical, and a fair proportion were verified at autopsy. Syphilitic cardiovascular disease is not included in this group. The coronary thrombosis cases were typical, with significant history, physical findings, electrocardiograms, and, in some instances, with autopsy; 19 developed auricular fibrillation, 13 developed right bundle-branch block, and 2 right bundle-branch block with later complete block.

TABLE V

Total autopsies	465	
Goiter heart, toxic		4
Pericarditis		
Acute fibrinous		3
Adhesive, chronic		3
Purulent		3
Endocarditis		
''Malignant''		2
Subacute bacterial		2
''Rheumatic''—acute*		7
''Rheumatic''—chronic		2
Aortic stenosis		2
Coronary disease		
Thrombosis		20
Thrombosis with heart rupture		1
Hypertensive cardiovascular disease		35
Arteriosclerosis		3
Syphilis of aorta		3
Aneurysm		1
Myocarditis		
Acute toxic		2
		96

*Not acute rheumatic fever, but acute endocarditis on the basis of old rheumatic lesions of the endocordium.

In the goiter group there were 144 patients who showed various degrees of effect upon the heart. Many simple goiters were seen which are not included.

In addition to these classified cardiovascular patients, 651 patients came for heart examination in whom no heart disease was found.

Hypertensive cardiovascular disease shows a high incidence, as in other localities. This term is used to include those cases with peripheral arteriosclerosis, retinal arteriosclerosis and aortic sclerosis as shown on physical and x-ray examination rather than to attempt to separate those with slight or doubtful arteriosclerosis from those with obvious evidence of arterial thickening. It is the commonest type of circulatory disease, resulting in cardiac, renal or vascular involvement.

In 465 autopsies at the Good Samaritan Hospital 96 showed cardiovascular lesions as the cause of death. They are grouped in Table V.

CONCLUSIONS

1. Heart disease as seen in the Pacific Northwest shows a low incidence of acute rheumatic fever (0.1 per cent, while in other localities the incidence varies from 5.8 to 0.2 per cent).

2. Hypertensive cardiovascular disease is the most frequent of all types (56 per cent).

3. Goiter, being endemic, shows an incidence of 6.1 per cent of medical patients producing cardiovascular symptoms severe enough to send the patient to the physician.

4. Hospital records, though of standard type for indexing, are entirely inadequate for statistical study as to etiologic and structural diagnosis of heart disease. They are satisfactory, however, as to incidence of cardiovascular disease as compared with total medical admissions. It is hoped that studies such as this may lead to more careful supervision of hospital records by medical boards or committees of physicians who will see to it that obsolete terms are eliminated.

5. Carefully studied patients in private practice with complete records offer a satisfactory basis for statistical study but may not give a true index as to incidence in relation to total population.

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(For discussion, see page 115.)

A CASE OF SINUS ARRHYTHMIA

With Pulse Slowing, Accompanying Each Second Normal Respiration*

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INTRODUCTION

JUVENILE sinus arrhythmia, consisting of acceleration of the pulse rate during inspiration and retardation during expiration is a common physiological phenomenon. Independence of the pulse irregularity and the normal respiratory cycle is less common but has been reported frequently. Our case, showing pulse slowing with each sec-

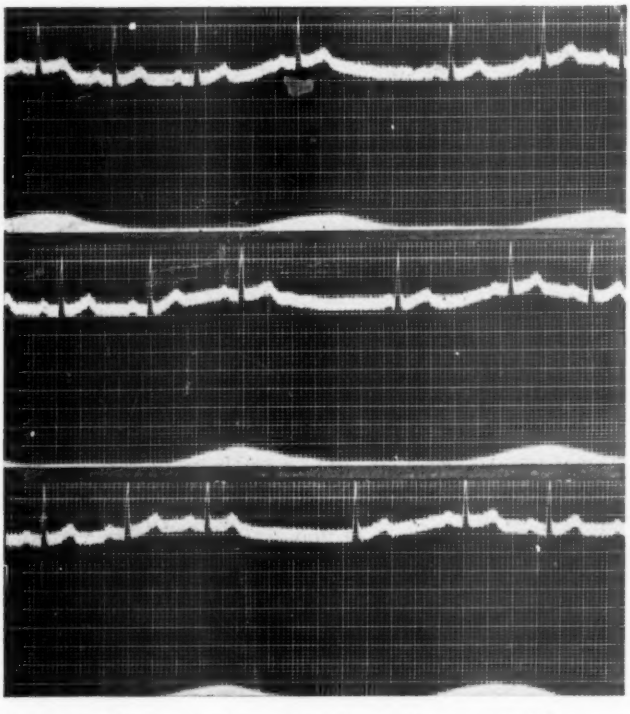

Fig. 1.—11/9/28—Continuous tracing. Normal respiration. Cardiac retardation with each second respiration. Ventricular escape.*

*The respiratory rate in all tracings was recorded on the electrocardiographic film as follows: The patient assumed a sitting position to one side of the film box, facing it. One end of a ruler was placed on his left chest anteriorly, with the other end in such a position as to cast a shadow on the film. A rise of level in the respiratory curve indicates inspiration. The method is sometimes inadequate in its representation of the depth of breathing. (cf. Fig. 6). The timing is fairly accurate. All electrocardiograms were taken in Lead II.

*From the Robinette Foundation, University of Pennsylvania Hospital.

ond normal respiration, does not fit into either of these two recognized groups. To our knowledge none like it has yet been reported.

CASE REPORT

G. M., aged twenty-four years, a white male student at the University of Pennsylvania, had suffered from frequent, prolonged winter colds for many years. He had experienced mild growing pains during his childhood, but had been otherwise well. In 1925 an enlargement of his thyroid was noticed, and shortly after that time, mild dyspnea and palpitation appeared and have persisted. In February, 1928, he had an attack of bronchopneumonia and entered the Students' Ward of

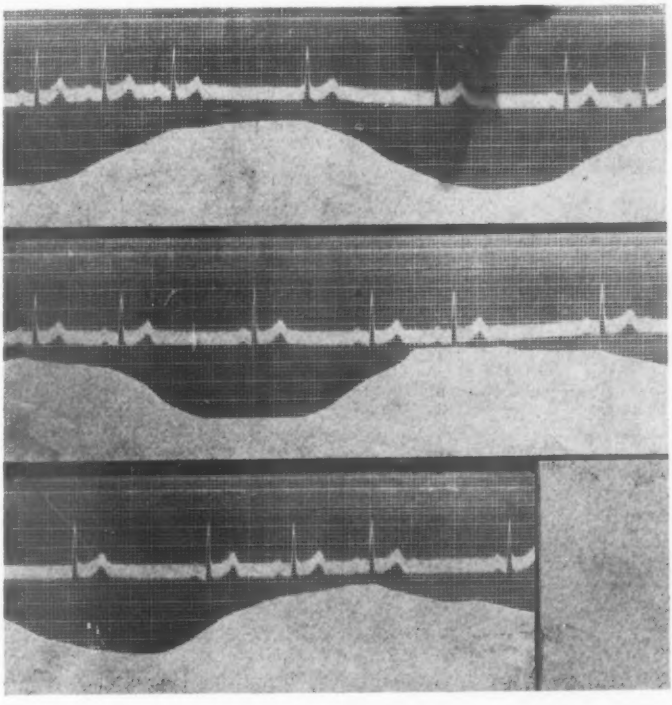

Fig. 2.—11/12/28—Continuous tracing. Deep respiration. Cardiac retardation with each respiratory cycle. Ventricular escape.

the University Hospital. During convalescence he became nervous, and on March 19, 1928, his basal metabolism was found to be plus 14 per cent. It rose in July to plus 36, and in September to plus 40. The last report of January 5, 1929 showed a basal rate of plus 24. During 1928 he gained weight.

On physical examination, the patient was found to be a powerful, well-built man. He stuttered slightly and appeared somewhat nervous. His thyroid was definitely, diffusely enlarged, but there was no thrill nor bruit. He had no tremor nor exophthalmos. By percussion, his heart was found to be slightly enlarged to the left. X-ray examination confirmed this. The sounds were of good quality without murmurs or accentuations. The blood pressure was 130/80 mm., the pulse 75, and the temperature and respirations were normal. No signs of congestion were found in the lungs, liver, or extremities.

His blood Wassermann was negative. His blood count was normal. His urine was negative. His phthalein and Mosenthal tests were normal. An x-ray of his teeth was negative, and a nose and throat examination on January 7, 1929, revealed no foci of infection.

The noteworthy finding in this patient was a marked cardiac arrhythmia in which slowing occurred with each second, or occasionally with each third normal respiration (Fig. 1). The tracing also showed ventricular escape when the heart rate was markedly retarded. During deep breathing, the cardiac slowing recurred with every respiratory cycle (Fig. 2). Ventricular escape was likewise seen. When the breath was held in deep inspiration, the heart assumed a regular rhythm and the P-waves disappeared, probably indicating a shift of the pacemaker to the junctional tissues (Fig. 3). Following this period of apnea, the 2-to-1 ratio did not reappear for some time (Fig. 4). When it did, the conditions depicted in

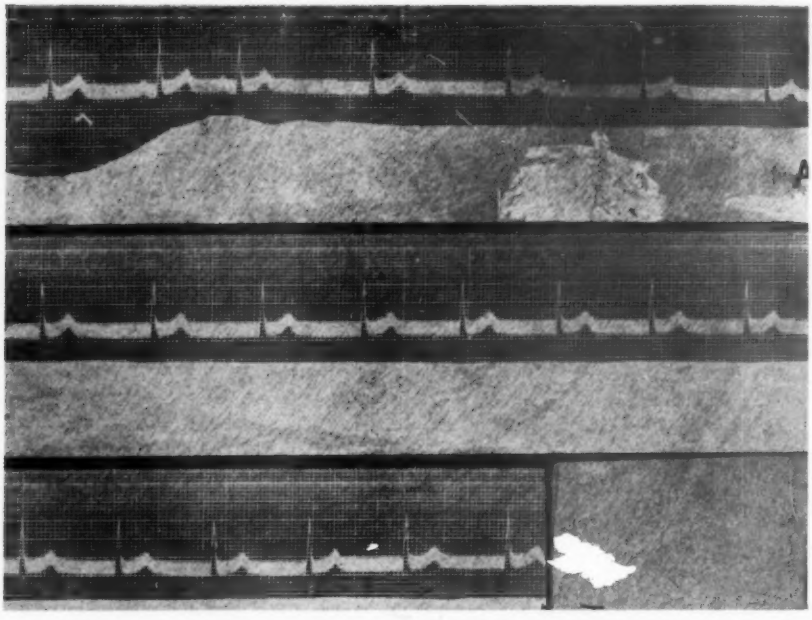

Fig. 3.—11/12/28—Continuous tracing except for the loss of ¼ inch at point marked A. Inspiratory apnea. No arrhythmia. P-waves disappear almost at once on the assumption of the expanded position of the chest.

Fig. 1 returned. After mild exertion (20 hops on the left foot), the rate rose to 140, remained at this height for three minutes, and then gradually slowed, with some irregularly recurring periods of retardation. In about five minutes the original 2-to-1 ratio was resumed. This seemed to be the normal state of affairs.

Two months later the patient returned for further study. He had his usual winter bronchitis, but no fever nor malaise. His electrocardiogram at that time showed no arrhythmia, but the heart rate had increased to 90 (Fig. 5).

Three weeks later, after he had improved but not completely recovered from his bronchitis, further tracings were made. During quiet breathing the rhythm was quite regular at a rate of 92, but during deep breathing it showed the same conditions seen in Fig. 2 (Fig. 6). During inspiratory apnea for 70 seconds it was perfectly regular with no disappearance of the P-waves, such as had been shown in Fig. 3. During expiratory apnea lasting forty seconds, the pulse at first was regular, but toward the end showed three periods of slowing (Fig. 7).

He came in once more two months after the last tracing with no further signs of his winter bronchitis. A tracing, taken at this time showed the same arrhythmia as that recorded in Fig. 1. We, therefore, consider this to be his normal state.

DISCUSSION

Sinus arrhythmia first attracted the attention of physiologists in 1860, when Ludwig suggested it to his pupil, Einbrodt, as a problem

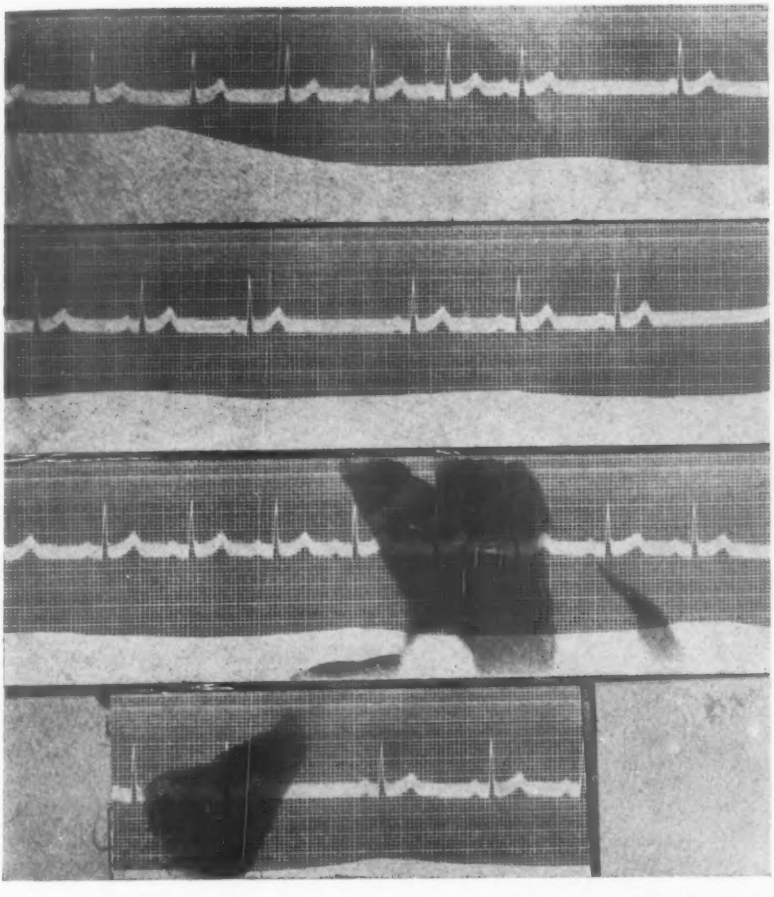

Fig. 4.—11/12/28.—Continuous tracing. Recovery period after Fig. 3. Irregular irregularity, not synchronous with respiration. P-waves appear almost at once on abandoning the inspiratory position.

for study. The earlier writers[1, 2, 3, 4, 5, 6] considered it entirely a question of alterations in the tone of the cardio-inhibitory center, because their experiments led them to believe that the accelerator system played no part in reflex regulation of the heart. Hooker[7] proved this to be an inadequate conception, in that he was able to produce reflex changes in the heart rate through the accelerator nerves after the vagi had been cut. This new knowledge, however, had little bearing

on the main disputed point, namely, the question as to the origin of the stimuli which change the tone of the cardio-regulatory centers. The hypotheses regarding this problem may be summarized as follows:

1. The stimuli arise in the lungs, due to changes in intrapulmonary pressure or in lung contour.[1, 2, 3]

2. They arise from the muscles of respiration.[5]

3. They overflow to the cardio-regulatory centers from the neighboring respiratory center, as each impulse is sent to the muscles of respiration.[6, 8]

4. They arise within the right auricle and great veins as a result of the rise of pressure within these vessels produced by the inspiratory aspiration of blood into the heart.[9, 10, 11, 12]

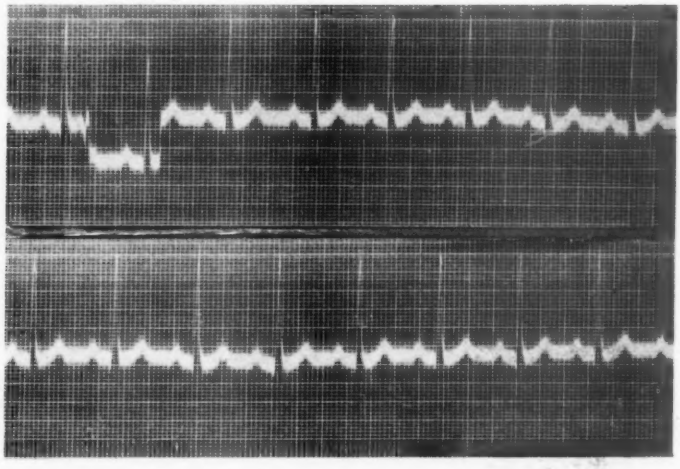

Fig. 5.—1/4/29—Continuous tracing. During bronchitis. No respiratory record. No arrythmia. Rate 90.

5. The possibility that rhythmic changes in blood P_H, due to respiration, can be the cause of sinus arrhythmia has been suggested by Sanderson,[13] but does not agree with our present knowledge of the subject.[14]

On account of its general characteristics and its behavior under the conditions imposed, the irregularity in our case almost undoubtedly belongs in the group generally known as "vagal" or "sinus" arrhythmias.* It differs, however, from the common juvenile type. The normal "youthful arrhythmia" (Mackenzie) seems dependent as a rule upon changes in either direction from what might be considered the basal rate of the heart, quickening during inspiration and slowing during expiration. If either of these is more pronounced, it is usually the former. Our patient's arrhythmia, however, seems almost entirely

*An additional bit of evidence, the effect of atropine was not studied, because the patient preferred not to have the drug administered, and because it was not thought necessary, since any type of tachycardia abolished the irregularity.

dependent upon a periodic slowing, usually during expiration, with very little, if any, inspiratory acceleration above what might be considered the basal rate of his heart. This fact is most definitely shown in Fig. 6.

Wedd[15] believes that this general class of arrhythmias is due to an imbalance between accelerator and vagus control of the heart, with periodic increased activity of the weaker of the two systems in an

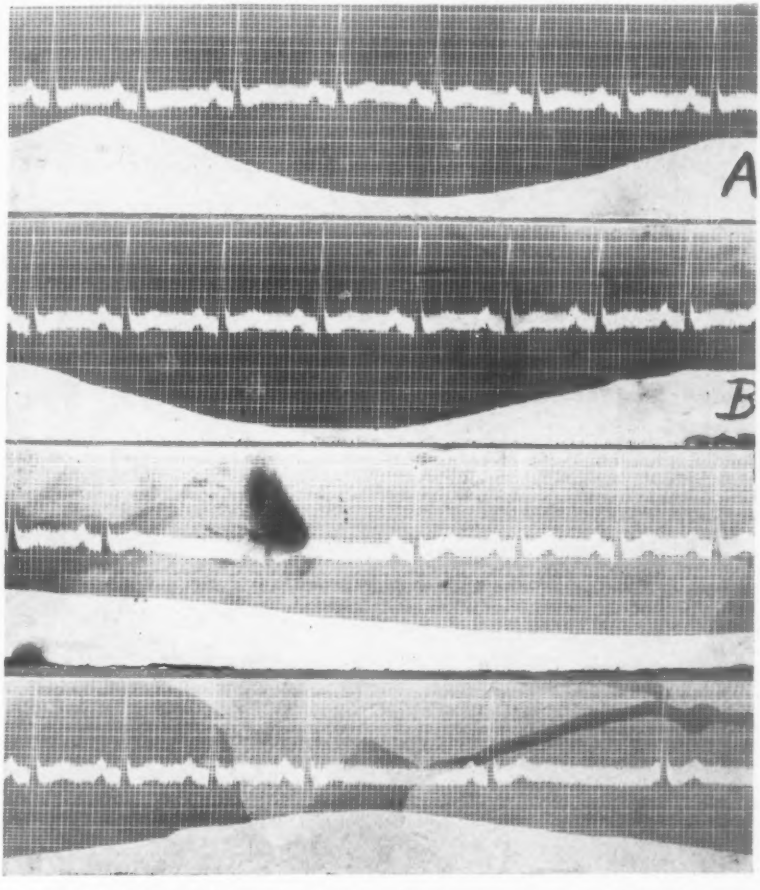

Fig. 6.—1/29/29—Continuous tracing. Toward the end of attack of bronchitis. Two normal respirations followed by two deep respirations. The respiratory record does not show accurately the depth of respiration. *A* shows the last normal breath, *B*, the first deep breath. No arrythmia with normal breathing. Arrythmia occurs during deep breathing.

attempt to bring about a more perfect balance. In the light of this hypothesis, our patient might be thought of as a case of sympathetic preponderance, possibly ascribable to his thyroid disease. The vagus, being the weaker of the two systems, periodically attempts to equalize the balance, but has greater difficulty in doing so during any period

of increased cardiac activity, such as that accompanying the attack of bronchitis (Figs. 5 and 6).

Wedd did not discuss the mechanism underlying this periodic vagus intensification, nor can we offer an adequate explanation for it in our patient. However, the following facts are brought out in our tracings. During inspiratory apnea the P-waves disappeared from the electrocardiogram. Their disappearance and reappearance followed quite closely upon the assumption and abandonment of the expanded position of the chest (Figs. 3 and 4). It is therefore possible, in our patient, that the inspiratory position stimulates the vagus, inhibiting the

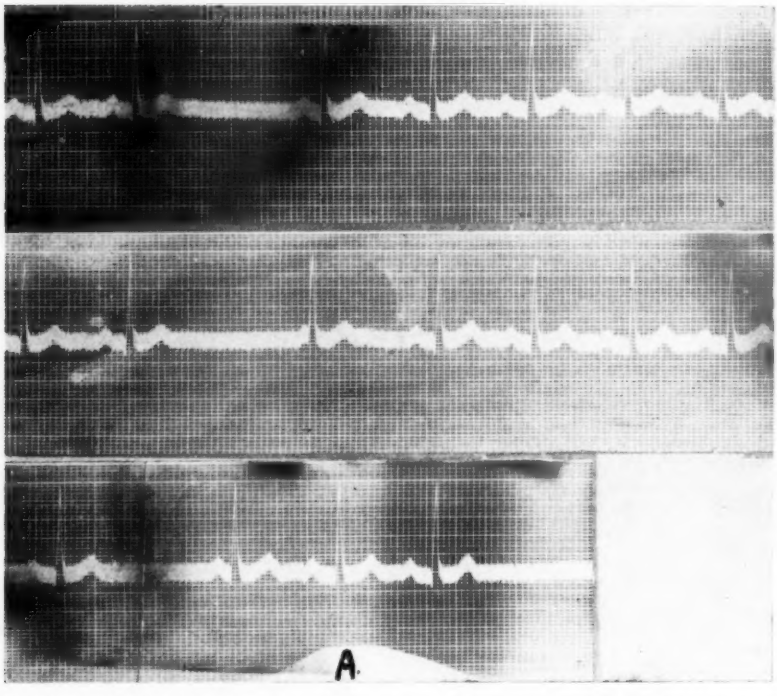

Fig. 7.—1/29/29—Continuous tracing. Toward the end of expiratory apnea. The respiratory curve does not show during apnea, but the resumption of respiration may be seen at *A*. Three periods of cardiac retardation in the absence of respiration.

sino-auricular node, thereby causing a shift of the pacemaker to the junctional tissues. If this is conceded, periodic vagal stimulation as a result of rhythmic assumption of the inspiratory position might be the factor causing the arrhythmia during respiration. The resulting inhibition, after a short latent period, appears during expiration. This latent period is shown in Figs. 3 and 4, at the beginning and at the end of inspiratory apnea.

The occurrence of the arrhythmia during expiratory apnea, however, necessitates the postulation of another source of stimuli, presumably extrathoracic, affecting the vagus center, when rhythmic stimuli from

the thorax cease. This may be some point in the central nervous system, as suggested by Fredericq.[6] Anoxemia, the result of apnea, which is known to increase cardio-inhibitory tone[14, 21, 22] may aid this secondary source of stimuli in producing its effect. During inspiratory apnea, the action of this subsidiary source of stimuli was prevented from becoming apparent, possibly because continuous vagal stimulation overshadowed it.

The 2-to-1 relation of cardiac arrhythmia and respiration is the most unusual and inexplicable feature of this case. Vagal arrhythmias independent of the normal respiratory cycle are seen fairly frequently.[16, 17, 19, 20] They occur as a rule in older people, but may appear in the young. According to Lewis[17] they usually become synchronous with the respiratory cycle when breathing is deepened. However, no case has been found in the literature similar to the one reported here. It suggests as its cause some cumulative factor, not strong enough to become effective with each quiet respiration. It was not due to change in the depth of alternate respirations since this was not present. It recurs too regularly for it to be the accidental coincidence of a periodic nonrespiratory cardiac retardation with each second respiratory cycle.

SUMMARY

A case is reported in which a hitherto undescribed form of cardiac arrhythmia appears. Under normal respiratory conditions retardation of the heart occurs with each second respiration. Simultaneous electrocardiographic and respiratory tracings are shown, and a discussion of the mechanism involved is presented.

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ELECTROCARDIOGRAPHIC ELECTRODES*

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THE most disturbing factor encountered by one doing technical electrocardiography is the proper application of electrodes and the resulting overshooting. Since the more or less universal abandonment of the solution electrode, there have been numerable types recommended, such as plates of various metals and the direct application of copper wire. These all require preparation of the skin before application, by heat, salt solution, acetone, scrubbing, etc., so as to reduce overshooting to a minimum.

In order to reduce overshooting, I have for the past six months used a pad known as "The Chore Boy," manufactured by the Metal Textile Corporation, Orange, N. J., made primarily for scouring kitchen utensils. This pad has woven

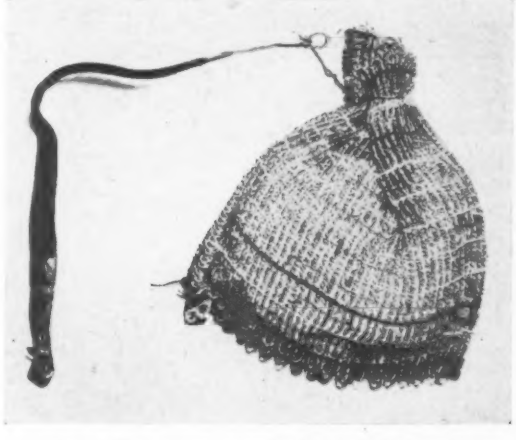

Fig. 1.—The pad prepared as an electrode.

through its loose mesh, fine strands of copper wire which are rough and resemble long turnings or shavings. The pad is made in the form of a mitt, having an opening at one end, by which it is filled with a small amount of cotton. This opened end is brought together and bound by a copper wire, which by an ordinary radio ground connection is connected with the lead wires of the electrocardiograph.

The pads are saturated with salt and moistened with hot water. Two of these are placed on the arms of a comfortable chair, while the third is placed on a rubber mat insulating it from the floor. The patient is required to remove the left shoe and stocking, preparation of the skin is unnecessary. The left foot and the palms of the hands are placed on their respective pads. Numerous electrocardiograms can be taken without rewetting the pads, but if the skin resistance is high, salt and water is again poured on the pads. The patient should be told to relax and be as quiet as possible.

Occasionally, somatic tremors or a traveling string is encountered. On these rare occasions the pads for Leads I and II are held to the wrist by rubber bands,

*From the Cardiological Departments of White Cross and Children's Hospitals.

made by cross sectioning an ordinary automobile inner tube. By this method overshooting has been completely overcome. It is probably due to the fact that the rough strands of copper shavings press deeply into the skin and make a more perfect contact than any other type of electrodes now available.

CONCLUSIONS

1. The use of these copper woven pads removes the necessity of skin preparation.
2. The electrodes are constantly in position and need not be connected for each electrocardiogram.
3. Overshooting has not been encountered, and standarization of the string is made easy by using this method.

Society Transactions

AMERICAN HEART ASSOCIATION

FIFTH ANNUAL SCIENTIFIC SESSION

JULY 9, 1929

The fifth annual scientific session of the American Heart Association was held in the auditorium of the Woman's Club, Portland, Oregon, July 9, 1929. The session was called to order at 2:10 o'clock by the president, Dr. William H. Robey of Boston, Mass.

DR. WILLIAM H. ROBEY.—Will the fifth annual meeting of the American Heart Association please come to order. Dr. Herrick has been upset because, owing to a change in the time of his clinic, he felt he could not come to this meeting. We regret that he cannot be here for the whole session, but as he is here now, we will ask him to try his lecture on us.

1. DR. JAMES B. HERRICK, Chicago, Ill.—**Coronary Occluson.** (For original article see AM. HEART J. **4**: 633, 1929.)
2. DR. T. HOMER COFFEN, Portland, Ore.—**The Incidence of Heart Disease in the Pacific Northwest.** (For original article, see page 99.)
3. DR. J. H. CANNON, Charleston, S. C.—**Clinical Observations on Syphilitic Occlusion of the Coronaries.** (For original article, see page 93.)
4. DR. IRVING R. JUSTER and DR. HAROLD E. B. PARDEE, New York, N. Y.—**An Electrocardiographic Study of Fifty Cases of Cardiovascular Syphilis.** (For original article, see page 84.)
5. DR. EUGENE S. KILGORE, San Francisco, Cal.—**The Problem of the Nervous Heart.** (For original article, see page 9.)
6. DR. ROBERT L. BENSON, Portland, Ore.—Exhibits of specimens of rupture of the heart due to cardiac syphilis in the Scientific Exhibit of the American Medical Association.

DISCUSSION

DR. WILLIAM H. ROBEY, Boston, Mass.—I want to say a word about Dr. Herrick's interesting paper. I think in cases with repeated attacks of cardiac pain small areas of scar tissue are found at autopsy resulting from occlusion of twigs of the coronary. That has been shown by Louis Gross and others. Occlusion of twigs is probably the cause of the attacks of angina pectoris, and they have also shown how one side by anastomosis will help the affected side of the heart.

I want to emphasize other symptoms replacing pain. I recently saw a man weighing 230 pounds who had always been perfectly well, but who had dyspnea in very much the way pain comes in angina pectoris. Walking along the street this patient could go only about a block when he would be forced to stop because of intense dyspnea. He has been given cardiac rest, the symptom has entirely disappeared, and he is able to walk without attacks of dyspnea.

Another important symptom is *nocturnal dyspnea.* A person may be free of symptoms throughout the day, yet during sleep when the heart quiets down there is a deficient blood supply to the muscle with a resulting anoxemia and the patient wakens with dyspnea. A prominent man in Boston went to a colleague of mine for a thorough examination. He was planning an extensive trip and was told that it was safe to take it, but he collapsed on the train, was examined at the Mayo Clinic and advised to return to Boston where he died three weeks after his arrival.

Now the doctor who made the first examination was a very careful man, but he had forgotten to ask about nocturnal dyspnea. This patient, once or twice a night for a year, had been wakened by air hunger, but he and his wife had neglected to say anything about it, and because the doctor had not thought to ask, a very interesting point had been overlooked.

In the milder cases of coronary disease, time and rest may establish the function of the thebesian vessels, thus furnishing an increased blood supply to the impoverished muscle.

As to the pain of coronary disease, it seems to me it must be within the heart muscle. Of course, we do not know just how the pain is produced, but I think there are certain reasons against the theory of the French school and also of Dr. Allbutt. I have always believed (as maintained by Mackenzie and others) that it is a spasm of the heart vessels, because there is an analogous pain in the muscles of the leg. A patient with arteriosclerosis is suddenly seized with pain in the leg; he rests a moment and the pain ceases. That was probably a spasm of the leg vessels, the muscles calling for more blood.

We have studied at the Boston City Hospital cases of gall bladder disease in which there has been a question of angina pectoris. In certain cases where there has been great doubt, we have recommended operation and have found that gall bladder disease did exist. It seems to me if there is any great doubt in a chronic case it is better to give the patient the benefit of operation. One man who was under my care for a number of years had shortness of breath and substernal oppression when walking from his office to his house, which necessitated his climbing a small hill. He had more or less indigestion at the same time and finally had a mild attack of jaundice. He was operated on and found to have a small, narrowed gall bladder. He recovered from the operation very well, but about eight weeks later had a hemiplegia with complete aphasia. About four months later he had a very definite attack of coronary occlusion with cyanosis, sweating and fever, and died in a few hours.

Dr. George Dock, Pasadena, Cal.—In regard to the matter of pain confusing other conditions, one of the best cases of left coronary occlusion I ever had was a doctor about 50 years old whom I saw when he was well and strong, but who had had this pain for some time, with dyspnea on exertion. To me his condition was perfectly clear, but he, an unusually keen and clear-minded country practitioner, insisted that he had cancer of the stomach. That was what he came to me for. We often discussed it in the time I saw him, and he insisted that his pain was exactly like that of cancer of the pylorus. It turned out, however, that he did not have cancer, but an obstructed left coronary.

Cases of syphilitic disease of the coronary are certainly among the most interesting of all. In many cases we have no doubt about the etiology. The most extraordinary one I ever saw was in a man 50 years old, who was brought into the hospital at Ann Arbor with advanced decompensation of long standing, and who died there. He was a very intelligent man and had some intelligent relatives with him who knew his history. At the age of twenty years he had a chancre followed by a period of anginal pain. The attacks were among

the most characteristic I have heard described, but while most patients with angina are obliged to keep still during the attacks, this young man, who had an ungovernable temper, when the attacks began would run around the room cursing and screaming, showing about as much muscular activity as a man in good health. He got over that attack, remained free from symptoms for a long time, but finally developed decompensation and died. Post-mortem examination showed that he had a healed infarct about 3 cm. in diameter, evidently of long standing. There were two features of interest in his case—the strangeness of his attacks of pain, and the fact that having had a severe lesion like that he recovered so completely. Of course that is not unique, but I thought at a meeting of this kind it might be worth while to add it to the comments that have been made.

Dr. Walter J. Wilson, Detroit, Mich.—Recently I had a case of *angina sine dolore* in a young man of 26 years, the presenting symptom being tachycardia, the auscultatory rate being 175, but on electrocardiographic examination we discovered a case of ventricular tachycardia and the rate 300. There was a leucocytosis of 31,200. There were no signs of decompensation. Under treatment of digitalis he improved, and the rate became normal in eight days, but when last seen he had right bundle-branch block. The use of quinidine was ineffective and the symptoms unpleasant.

I think that in many of these cases, emphasis should be laid on electrocardiographic examination, which, in the vast majority of cases, will elucidate the case.

Within the last few months, I have seen a young negro 18 years old, who at 14 years developed syphilis from venereal contact and now has a marked aortic insufficiency. In some cases, years do not have to elapse before sufficient damage is done to the aortic valve to cause insufficiency.

Dr. B. O. Raulston, Los Angeles, Cal.—This seems a proper time to call attention to a report made by Dr. Fitz of Boston of a case, a woman of middle age, a school-teacher, who had clinically a typical angina, and who in the course of a study to determine whether or not she might have disease of the gall bladder was given iodides intravenously. She developed the classical symptoms of coronary occlusion and died. Another thing that is somewhat surprising is that we do not hear anything about the possible effect of thrombosis of the small twigs of the coronary producing acute changes and then chronic changes that may account for many of the irregularities of the heart. A good deal of work has been done on that subject in France and England, and I feel that this may be the true basis for irregularities which we attempt to explain on other grounds.

Dr. Leslie T. Gager, Washington, D. C.—The embolic phenomena of coronary thrombosis—those which we have been accustomed to call embolic, and which Dr. Herrick suggests may be due to thrombosis in situ—are of increasing interest. I should like to cast my opinion in favor of the embolic theory, for the reason that in a number of carefully studied patients I have found no evidence of infection, either local or general. In one of the most recent patients, a man of 56 years, who had had a coronary thrombosis eighteen months previously, the first symptom was pain in the right arm which lasted a few minutes. Two weeks later during the journey to the hospital he had pain in the right leg lasting several hours. During this attack he had no fever, and his leucocytes on admission were 7000. Subsequently he had pain in the lower abdomen, without fever, for three days, next a cerebral accident with a left hemiplegia. This was not accompanied by fever and also cleared up in the course of several days. Then after another week—this was six weeks after the initial pain in the arm—he had severe, agonizing pain in the upper abdomen, began to vomit dark red blood, and died

after twenty-four hours. The post-mortem findings confirmed the clinical diagnosis of coronary thrombosis with a mural infarct in the left ventricle, and hemorrhagic infarction of the entire ileum. In the colon there was evidence of regression of an area of hemorrhagic infarction which was considered the cause of the lower abdominal pain two weeks before death. In the brain no evidence in the right cerebral vessels could be found to account for the hemiplegia on the left side. In other words, a series of accidents had occurred, which in the arm and leg in the brain left no traces; in the large intestine, changes which were resolving, and in the small intestine, the acute terminal phenomenon of arterial occlusion. The embolic theory, with multiple fragments being cast off at intervals from the intracardiac thrombus, would better fit the facts in such a case, it seems to me, than the assumption of thromboses in these several sites.

DR. ROBEY.—I am glad to have Dr. Wilson speak of tachycardia. I think it is quite as important a symptom as dyspnea or pain. I have recently seen a physician of 62 years who had attacks of paroxysmal tachycardia. The physical examination was not important, but an electrocardiogram taken after the third attack showed a downward deflection of T_1 and T_2 with concavity of the S-T interval. This we find in cases of coronary thrombosis, and I made that diagnosis notwithstanding the absence of pain. Fred Smith found that after ligation of a coronary branch in dogs a similar electrocardiogram was obtained, but in a few months there was a return to a normal T. An electrocardiogram of my patient taken several months later was similar to the first.

DR. GAGER.—The early diagnosis of cardiovascular syphilis is a problem which is of great importance to all of us. On the Atlantic seaboard, as rheumatic fever declines in incidence going from north southward, syphilitic heart disease and hypertensive-arteriosclerotic heart disease increase. The differential diagnosis of these two types is often exceedingly difficult; therefor, if we can find in the electrocardiogram definite evidence of syphilitic myocardial involvement, it is an extremely valuable aid. Personally I have had a great deal of difficulty clinically and with the electrocardiogram in making this differentiation. For example, I have recently seen a patient in whom a six-foot plate showed dilatation of the aorta, so well demarcated that the roentgenologist was willing to make a diagnosis of aneurysm. At post-mortem, however, the lesions were apparently entirely arteriosclerotic. In other patients with undoubted clinical syphilis, there have been seen those changes which Dr. Juster has shown in the T-wave. I am thinking of a patient I saw a week ago in whom necropsy showed, in addition to aortitis and aortic insufficiency, patent coronary orifices, extensive fibrosis of the myocardium, coronary sclerosis with areas of thrombosis. In such cases the electrocardiographic evidence so far has seemed to be on the side of arteriosclerotic heart disease without respect to any specific etiology or to pathognomonic structural changes.

DR. AUDLEY O. SANDERS, Palo Alto, Cal.—Dr. Juster's paper has brought to mind a problem that came to our group a few weeks ago. A young man came to us having had a diagnosis of syphilis and giving a history of a four-plus Wassermann in 1923. Later Wassermanns, both blood and spinal fluid, had been negative. The patient denied luetic infection. Our blood and spinal fluid examinations were negative. The electrocardiogram was not definitely abnormal. The x-ray, however, showed a very marked dilatation of the ascending aorta. With this and the questionable history of lues we were inclined to a diagnosis of luetic aortitis with dilatation. This patient had another condition which led to his death. At autopsy we found, to our surprise, no evidence of luetic aortitis but a constriction of the arch of the aorta with marked dilatation of the ascending aorta proximal to that point. The autopsy diagnosis was that of a congenital condition of the aorta.

DR. JUSTER (closing).—This observation of the abnormality of the T-wave was made four years ago. Of course it took quite a while to collect these cases, but we have found that we could always use it as a diagnostic point. Quite often you see a patient in the thirties with aortic insufficiency with a positive Wassermann and a history of rheumatism, and the question arises—is the etiology syphilis or rheumatic fever? By using this method of the inversion of the T-wave, provided no digitalis has been given, we have been able to secure great help in the diagnosis of syphilitic aortic insufficiency. In watching some of these cases develop this inversion, increasing severity of symptoms was noted. In view of the fact that the T-wave inversion is common in valvular cases, repeated electrocardiograms may be an aid in determining whether or not our treatment of early syphilis will prevent the development of the cardiovascular lesion. We know we get a definite result in early lues, but we have not followed these cases long enough to say whether we have prevented the development of cardiovascular disease. The cases that develop the T-wave inversion do very poorly, and of course the ultimate outcome is usually fatal. We find that treatment after this T-wave abnormality has occurred is of no help whatever.

Department of Reviews and Abstracts

Selected Abstracts

Lightwood, R. C., and Davies, M. Llewelyn: A Clinical Study of Acute Rheumatism. Brit. M. J. 495, September 14, 1929.

This report is based on work at the Rheumatism Supervision Center, Royal Borough of Kensington. The primary object of this is to prevent relapses and to care for those children whose hearts are already involved. In the latter group of children these two objects are inseparable. The functions of the rheumatism supervisory center as outlined should be: first, to make use of all the measures available for preventing relapses in rheumatic children, and second, to supervise the after care of children already the victims of rheumatic heart disease.

The lines on which recurrences of rheumatism may best be prevented are: one, correction of unsuitable environmental conditions; two, attention to general health; three, treatment of diseased tonsils; four, education of the parent in the correct management of the rheumatic child.

Hill, N. Gray, and Allan, Mary: The Rheumatic Type. Brit. M. J. 499, September 14, 1929.

A general study of 562 rheumatic children and 536 other patients under similar conditions admitted to the hospital for study leads to the conclusion that there is no type as regards complexion, color of hair, eyes, etc., that can fairly be described as the rheumatic type of child. The total division of complexion is about equal between fair and dark children. There also was no evidence that the skin of the children of the rheumatic group was more dry than among the nonrheumatic patients.

White, Paul D., and Mudd, Seeley G.: Observations on the Effect of Various Factors on the Duration of the Electrical Systole of the Heart as Indicated by the Length of the Q-T Interval of the Electrocardiogram. J. Clin. Investigation **7**: 387, 1929.

A study is here reported of the measurements of the Q-T interval made on carefully selected electrocardiographic plates with the help of the Lucas comparator in 213 individuals, of whom 50 were normal to act as controls and 163 were abnormal subjects to illustrate the effect of various pathological conditions. The prime factor influencing the duration of both mechanical systole and the Q-T interval of the electrocardiogram has been found to be the heart rate. The faster the rate, the shorter the duration of the Q-T interval, although at faster rates the relative proportion of the heart cycle made up by systole steadily increases.

The measurement of the duration of the Q-T interval of the electrocardiogram is apparently of little or no clinical value from the study of these cases.

Gray, S. H., and Aitken, Louis: Late Gross Lesions in the Aorta and Pulmonary Artery Following Rheumatic Fever. Arch. Path. **8**: 451, 1929.

It is the purpose of this paper to report the late gross lesions that were discovered in the aorta and pulmonary artery of the rheumatic hearts examined in four patients. In one of these there was an aneurysm of the aorta in which all

the evidence pointed to a rheumatic origin. The criteria of Pappenheimer and von Glahn for the establishment of the diagnosis of rheumatic fever has been adopted in selecting cases for the study. All four patients were adults, ranging in age from 20 to 47 years of age. The late lesions persisted in scarring of the media and thickening of the intima.

The authors believe that the gross lesions occur as a result of a confluence in a localized area of the small flame-shaped scars occurring during the acute stages of the disease. It was possible in each instance to determine that syphilis had not been present.

The formation of aneurysm appears to be the result of a break in the aorta with a subsequent dissection of the aorta along the lower part of the media.

Small, James Craig: The Rôle of Streptococci in the Rheumatic Diseases. J. Lab. & Clin. Med. **14: 1144, 1929.**

The rôle of streptococcus in rheumatic disease is discussed and a hypothesis embracing a dual nature of its pathogenesis is offered as follows:

The specific toxin of a streptococcus is suggested as operative in the production of the "destructive" and "proliferative" types of lesions in rheumatic fever, while the patient's hypersensitization to an allergen associated with the protein of streptococci is presented as concerned in producing the "exudative" lesions. The specificity of this sensitizing substance or allergen is not dependent upon the type of streptococcus supplying it.

Three types of reaction in patients are described as "exudative lesions" best exemplified by the acute arthritis; the "proliferative" lesions typified in their purest form in the subacute progressive cardiac lesions in the heart; and the "destructive" lesions best seen in the heart muscles of patients who have died early in an attack of rheumatic fever attended by an overwhelming clinical toxemia. This lesion amounts to actual sterile necrosis of the muscle fibers occurring in areas of a considerable extent.

The author believes that the destructive lesion is brought about by high concentrations of a specific endotoxin derived from a particular group of streptococci; the proliferative lesion because of a stimulating effect of weaker concentrations. The exudative lesion appears when the patients begin to develop immunity to the specific toxic factor and is brought about by the establishment of a condition of hypersensitiveness which is a manifestaiton of the Arthus phenomenon. These lesions may be accounted for on the basis of bacterial "protein" specificity and not on the basis of bacterial group or type specificity.

The author proceeds to describe chronic arthritis as an allergic disease, the hypersensitive state of the patient being due to this allergen contained in streptococci without regard to a particular type.

Levine, Samuel A., and Brown, Charles L.: Coronary Thrombosis: Its Various Clinical Features. Medicine **8: 245, 1929.**

The author makes the following considerations as the result of an analysis of the clinical features of 145 cases of coronary thrombosis and the pathological data of 46 of these.

1. Angina pectoris generally precedes attacks of coronary thrombosis but there were a few instances in which it was quite clear that the patients not only had no angina but no evidence of any important preexisting disease could be made out.

2. Coronary thrombosis frequently developed in long standing mild diabetics, but because the age incidence was the same in diabetic as in nondiabetics it would seem that the diabetes merely indicated the type of individual who would develop coronary disease rather than that it had any causative relation to it.

3. Hypertension was present in the great majority of cases but in some it was quite definitely known that the blood pressure was normal before the attack. Arteriosclerosis was a very variable finding. In some it was strikingly limited to the coronary arteries.

4. Syphilis was found to be a very rare cause of coronary thrombosis, and other infectious diseases seemed to have very little etiological significance.

5. Hereditary factors, although extremely difficult to analyze, were found to be most important especially in those patients having coronary thrombosis at a comparatively young age. Possibly as a part of the hereditary factor there seems to be a certain physical type of individual who is more apt to develop this disease. The type is that of a well set and strong individual, somewhat overweight, whose limbs and especially the forearms are round rather than flat. He generally has been quite active physically, either in sport or at work.

6. The average age in this series was 57.8 years. There were 111 males and 34 females. The marked disproportion in the sexes cannot be easily explained but brings up the possible relationship of physical work and tobacco to coronary disease, both of which factors are more prominent in the male than in the female.

7. The typical clinical picture of acute coronary thrombosis was discussed in detail. In addition certain atypical features were emphasized that are commonly overlooked and which are important in making a proper diagnosis. The pain was found to vary from a slight discomfort in the chest to the most terrific agony, and varies in the location from the upper abdomen to the upper sternum and throat. There were not infrequent cases that were entirely painless. It was emphasized that in some instances the entire picture resembles very closely an acute surgical abdomen. Although there customarily was a fall in the blood pressure with the attack, in some instances this did not occur.

Fever and leucocytosis developed early in most cases but there were rare exceptions. The temperatures must be taken rectally as the mouth readings were frequently normal when an actual fever was present. The important features on examination were the appearance of shock, the distant heart sounds, gallop rhythm, the development of various irregularities in the rhythm of the heart, occasionally a pericardial friction rub, râles in the lungs and sometimes an engorgement of the liver.

Certain changes in the electrocardiograms were found to be invaluable as aids in diagnosis, both during the early days and also in the later weeks following the attack. Besides those electrocardiographic changes that have previously been described, attention was called to the development of a prominent Q wave in Lead III in many of these cases.

The urine was frequently found to contain sugar and evidence of renal damage such as albumin and casts. At times there was marked oliguria or a suppression of urine. These findings generally were transient.

Both the types of death and the types of recovery, because of their variability, were analyzed and for the most part they were found to fall into fairly definite groups. This enabled one to predict somewhat more clearly the course of the disease.

8. The important conditions that at times had to be considered in differential diagnosis were an acute surgical condition of the abdomen, angina pectoris, pneumonia, diabetic acidosis, and finally so-called chronic myocarditis. The proper diagnosis in most cases is possible, although to make it in some, all our methods of study including electrocardiograms may be necessary.

9. The criteria for prognosis in individual cases were found to be most unsatisfactory. In general about 50 per cent have an immediate recovery. No single feature seemed to be reliable as indicative of a good or poor prognosis. Apparently mild cases occasionally died and very severe ones recovered. Slight differences in the mortality were found when certain factors were analyzed such as age, sex, the

development of pericarditis, and auricular fibrillation. Ventricular tachycardia and heart block seemed to have a greater mortality than the average. Even the type of change in the electrocardiogram had no influence on whether the patient would recover or not.

10. The question of treatment for the present must be based partly on theoretical grounds as there are no data available to compare the end-results of one régime with those of another. The acute and rapid character of the disease often makes our deductions as to therapy fallacious, because frequently many drugs are given in a short time and it is difficult intelligently to appraise the proper value of any single one. A proper understanding of the pathological process going on during coronary thrombosis will help to some extent in rationalizing our therapy. Certain features in treatment were discussed which we consider may prove life saving in occasional cases.

11. A careful pathological study of 46 of these cases was made. Apart from the ordinary findings some interesting correlations with the clinical features were uncovered. It was found that not infrequently a thrombus formed in the right ventricle as well as in the left as a result of a thrombosis of the left coronary artery. This happened when the interventricular septum was involved. There were two painless cases in which the right coronary artery was thrombosed. In nine cases rupture of the ventricle occurred. The most frequent artery involved was the left descending coronary and the favorite site of the thrombus formation was about 2 cm. below the bifurcation with the left circumflex coronary artery.

Stewart, Harold J.: The Effect of Exercise on the Size of Normal Hearts and of Enlarged Hearts of Dogs. J. Clin. Investigation 7: 339, 1929.

The authors have investigated this subject, using dogs in which defects of the mitral valve have been made by operation and in which enlarged hearts have in consequence developed. There were no signs, however, of heart failure. Control animals were included in the study. The dogs had been the subjects of other experiments and had been operated on two to three and one-half years ago.

The dogs were first trained to run on a treadmill and after preliminary training the effect of running on the size of the heart was investigated by means of x-ray photographs of the heart. The dogs were allowed to run only as long as they did so voluntarily usually from twenty-five to sixty minutes. It was found on examining the x-ray photographs that the size of both normal and enlarged hearts always decreased. There was no evidence of dilatation of the heart. The heart muscle of these animals is presumably free of myocardial disease since no infection had been introduced in the experimental conditions. These observations bear out the general assumption that myocardial disease is necessary for the development of heart strain, dilatation and cardiac collapse.

Boas, Ernst P., and Weiss, Morris M.: Heart Rate During Sleep as Determined by the Cardiotachometer. J. A. M. A. **92:** 2162, 1929.

By means of the cardiotachometer, the authors have observed the pulse rate over long periods of time during sleep, rest and activity, in both normal and abnormal human subjects. This study has shown the tremendous variability of the normal heart rate as well as its marked reduction and relative stability during sleep.

They have also noted in patients with exophthalmic goiter active myocarditis and at times mitral stenosis that the drop in rate during sleep is greatly diminished. They point out that this increased rate during sleep may serve as a valuable aid in diagnosis especially in differentiating organic heart disease from those patients with a neurogenic tachycardia.

Hart, A. P., and Silverthorne, L. N.: A Case of Acute Bacterial Endocarditis. Canad. M. A. J. 21: 305, 1929.

A case of bacterial endocarditis is reported embodying some extremely interesting findings in a female child thirteen years of age. Encephalitic symptoms were the outstanding features in the case when the child was admitted to the hospital. Streptococcus viridans was recovered from the culture of the spinal fluid. Petechial hemorrhages, palpable spleen, enlargement of the heart with a systolic murmur and the recovery from the blood of streptococcus viridans were the main clinical findings. Autopsy revealed petechial hemorrhages in the heart, brain, skin and mucous membranes with embolic manifestations in the heart and brain.

The patient had had measles, scarlet fever and whooping cough and two attacks of rheumatic fever in the last two years of her life. Because of the attacks of rheumatism, she had been kept at home though apparently well. There were no evidences of rheumatic carditis at autopsy. The valves were thickened and there were fresh vegetations on the margin. Microscopically the picture of the heart as a whole was that of bacterial endocarditis engrafted on an old rheumatic condition.

Hanzlik, P. J., and Wood, D. A.: The Mechanism of Digitalis-Emesis in Pigeons. Jour. Pharmacol. & Exper. Therap. 37: 67, 1929.

The emesis of digitalis has been studied in pigeons with the view of determining the seat of action by physiological and pharmacological methods and also according to the distribution of the drug in the body. The results obtained on pigeons indicate that the seat of emesis is peripheral in origin, the action consisting predominately of a vagus-reflex mediated through the local irritant action of the digitalis concentrated in the liver, other abdominal viscera not being excluded. The seat of emesis is not in the heart. It also seems that the seat of emesis is peripheral in origin and not central.

The following results support these conclusions: Digitalis is chiefly concentrated in the liver as compared with the blood, heart and lungs and the liver shows a comparatively greater sensitivity to digitalis than the peritoneum. Intraperitoneally, chemically unrelated irritants are as effective as digitalis in causing emesis but intravenously the unrelated irritants do not cause emesis and digitalis does. Peripherally acting emetics act on intravenous injection as does digitalis, whereas several typical medullary stimulants and centrally acting emetics do not cause emesis in pigeons, thus indicating the peculiar rôle of digitalis as a systemic emetic without direct stimulation of the central mechanism.

Of the autonomic nerves the parasympathetics are indispensable to the emesis since vagotomy prevents it.

Swetlow, George I.: A Clinicophysiologic Study of the Pathway of Pain Impulses in Cardiac Disease. Am. J. M. Sc. 178: 345, 1929.

This paper is presented to correlate the previously observed clinical findings in patients complaining of severe cardiac pain with the information obtained by others through animal experimentation. The clinical observation showed that subjectively the patient was relegated to areas of the skin which were supplied by nerves arising between the eighth cervical and seventh thoracic spinal segments. These very same skin areas to which the patient subjectively referred to pain were hypersensitive to protopathic tests. These findings indicated that the pain impulses were passing through the rami communicantes and ganglia which were found between the eighth cervical and seventh thoracic spinal segments. The indifferent operative results ensuing from procedures upon the cervical sympathetic chain and

other nerves in the neck indicated by their failure to ameliorate the pain that the painful charges were not passing through these structures in their course to the sensorium. A review was made of 41 patients suffering from severe cardiac pain who were treated by paravertebral block. The gratifying results obtained seem to suggest that these rami communicantes and ganglia are the true conveying pathways of the pain impulses. The résumé of the animal experimentation supports the clinical observation.

Cowan, John, and Faulds, J. Steven: Syphilis of the Heart and Aorta. Brit. M. J. 285, Aug. 17, 1929.

The authors have studied a series of 390 cases presenting naked eye signs of cardiovascular disease and have found 60 of these cases to be syphilitic in nature. The various pathological lesions of syphilitic heart disease are discussed and correlated with the various clinical data. Under treatment the authors believe that the true treatment of cardiac syphilis is preventive, the successful treatment of primary illness and that cardiac syphilis is the result of an error on the part of the patient or his medical adviser.

Ernstene, A. Carlton: Observations on Coronary Thrombosis. Am. J. M. Sc. 178: 383, 1929.

Three cases of coronary thrombosis with recovery and six cases terminating fatally have been studied. The recovered patients have been under observation for from one to about four years after the attack and all at present are enjoying satisfactory health.

Soon after the occurrence of coronary thrombosis there is commonly a marked reduction below normal of the vital capacity of the lungs. This observation is of diagnostic value because many patients at this time show few peripheral signs of myocardial failure. The conspicuously small cardiac contractions observed fluoroscopically after coronary occlusion are likewise of diagnostic assistance.

Progressive rise in blood pressure and vital capacity and increasing cardiac pulsations observed fluoroscopically are favorable prognostic signs. Conversely, falling blood pressure after the first few days, decreasing vital capacity and failure of the cardiac contractions to show improvement on fluoroscopic examination are unfavorable signs. Symptoms and signs indicative of extension of infarction are of very serious import.

The early recognition of coronary thrombosis is of importance because with proper management the patient may recover and live for years.

Chandlee, Gertrude Jackson, and Burvill-Holmes, E.: Clinical and Roentgen Ray Findings in the Study of the Heart and the Great Vessels. Am. J. M. Sc. 178: 364, 1929.

The authors have studied 100 cases of heart disease of different types by physical examination, electrocardiographic records and roentgen ray examination. They believe that roentgen ray study helps to demonstrate the functional efficiency of the heart in respiration, the degree of pulsation and relative changes in the various areas of the heart and vessel walls and that the classification of hearts that are not normal as inspiratory or expiratory in type is explanatory and a functional classification of value. They discuss the effect of forced inspiration and expiration on the appearance of the heart in fluoroscopic examination.

The authors believe that the change in shape and position and measurements of the heart when these two observations are compared is of assistance and value in

establishing diagnoses of heart lesions and especially pointing out abnormal physiological function. They discuss the appearance of the heart silhouette when various pathological lesions are present.

Ackermann, W.: The Treatment of Tuberculous Pericarditis With Effusion by Injection of Air and Lipiodol Into the Pericardial Sac. Am. Rev. Tuber. 20: 236, 1929.

Report is made of a patient aged 48 with tuberculous pericarditis treated by repeated aspiration of the fluid of pericardial effusion. In all twenty aspirations were done, seventeen of which were followed by air injection into the pericardial sac and once by injection of lipiodol. Notwithstanding the various abnormal conditions of the pericardium the heart itself appeared normal in size throughout.

The author states that the performance of artificial pneumopericardium is without damage and discomfort, that the injection of air gives greater relief than does aspiration of the air alone, and that the presence of air delays the reformation of exudate. By holding apart the two layers of the pericardium the friction rub and the formation of adhesions are prevented. On x-ray examination, the air in the pericardium assists one to see how completely the exudate has been evacuated.

Cookson, Harold: A Case of Cardiac Syphilis With Ventricular Aneurysm. Brit. M. J. 94, July 20, 1929.

The author reports a case of aneurysm of the ventricle occurring in a woman aged 40 who had epigastric pain with signs of congestive heart failure preceding death by two years. A clinical diagnosis of aortic syphilis had been made.

Autopsy showed three aneurysmal sacs on the posterior wall of the (L) ventricle, the walls of which were of fibrous tissue containing considerable deposits of fibrin. All the valves, the aorta and the coronary vessels were normal. Microscopic section showed necrotic tissue banished by these fibrous tissues, the rest infiltrated by plasma cells and lymphocytes, the plasma cells predominating. The vessels surrounding the degenerative area showed marked obliterating changes and in some cases, the lumen was occluded. The condition had been present no doubt many years.

Giraldi, J. J.: The Histology of the Aortic Wall in Acute Rheumatism. Bristol M. Chir. J. 46: 145, 1929.

Five cases of rheumatic fever among four children and one man aged sixty have been studied. Lesions of a distinctive character have been found in the aortic wall in all of them. These lesions consist of areas of subacute inflammations around a small vessel of a nature similar to those found in the pericardium and other tissues in cases of rheumatic fever.

Yater, W. M.: Congenital Heart Block; Report of a Case With Incomplete Heterotoxy. Am. J. Dis. Child. 38: 112, 1929.

An infant was found to have a slow pulse at birth and an electrocardiogram showed complete auriculoventricular dissociation. The infant's pulse rate had been noted to be slower than the maternal rate before birth. There was cyanosis and other congenital malformations. Death occurred on the eighteenth day. Autopsy showed complete auriculoventricular dissociation. The infant's pulse rate had been position and relative to each other. Histological study showed a break in the continuity of the bundle of His and the author was unable to identify the right branch of the bundle of His. The electrocardiogram showed no sign of right bundle-branch block.

Book Reviews

PRINCIPLES AND PRACTICE OF ELECTROCARDIOGRAPHY. By Carl J. Wiggers, M.D. St. Louis, 1929, 226 pages with 61 illustrations, The C. V. Mosby Co.

One method through which our knowledge of cardiac disease has been vastly increased during the past twenty years is by the use of instruments of precision, and of these latter none has given more definite information than the electrocardiograph. As a result there is a constant demand by physicians for postgraduate instruction in electrocardiography. The author of this volume, Professor Wiggers, has for years been giving courses in this field both at Cornell and at Western Reserve University. The use of the electrocardiograph, like the basal metabolism apparatus, has now become so extensive that personal instruction for all is no longer possible, and a book simply written is required for physicians and technicians who wish to instruct themselves in the theory and practice of electrocardiography. This book fulfills the need admirably.

The material of the book divides itself quite naturally into three parts, and of these the first part is the most difficult to present and the most valuable for the beginner. For here is explained in a clear and lucid manner the fundamental principles of electrocardiography and the physics of the galvanometer; finally there is an impartial review of all the important models of electrocardiographs available at present. Anyone working in this field is constantly asked to give his opinion on the relative merits of the various instruments on the market, and although the author quite properly does not make any specific recommendations, the reader obtains the necessary information about each model which should enable him to make his own selection according to his particular needs. This part on the instrument as such and directions for its use are especially helpful.

The second section describes the normal electrocardiogram and attempts to explain the meaning of the various curves. In this part the chapters on the significance of the electrical deflections are particularly well done, as it is most difficult to make this subject clear to medical students who have received no training in advanced physics or in electrophysiology. The author then proceeds to take up the usual abnormal records in logical sequence, and to correlate these abnormal electrocardiograms with the pathological physiology of cardiac disorders.

The third part places the reader in the position of a cardiologist who has to read the graphic records of clinical cases and interpret them.

This is analogous to the case system now so successfully used in medical and law schools. In each case there is first a description of the electrocardiographic findings, then an electrocardiographic diagnosis and the reasons therefore, then a brief description of the salient features of the disorder, with the relation to allied conditions (e.g., auricular flutter, impure flutter and fibrillation) and finally suggestions as to treatment.

The book is especially strong in the technic, in the instrumental aspect of the subject, and in the dynamics of the circulation—in those branches of the subject in which the author is an authority. The morphological aspects, e.g., the morbid anatomy as correlated to the electrocardiogram, are less emphasized, possibly because the author has had less personal experience in this field, possibly because he feels that we cannot speak with the same precision of the pathology of the heart as related to the electrocardiogram as we can of the physiology.

The frontispiece, a delicate and well-deserved tribute to the father of this science, contains an excellent portrait of Willem Einthoven.

As one reads this book, one cannot help remarking how much more definitely now than formerly many aspects of the whole subject can be presented to the beginner.

B. S. O.

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